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Journal of the TENNESSEE MEDICAL ASSOCIATION

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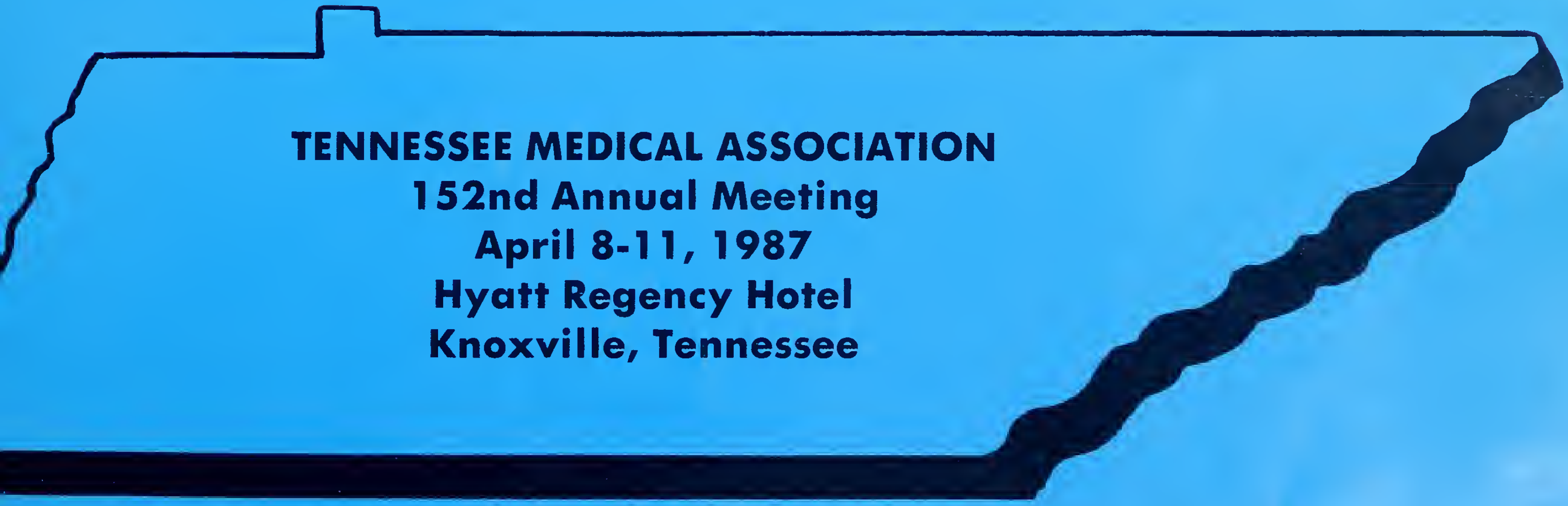

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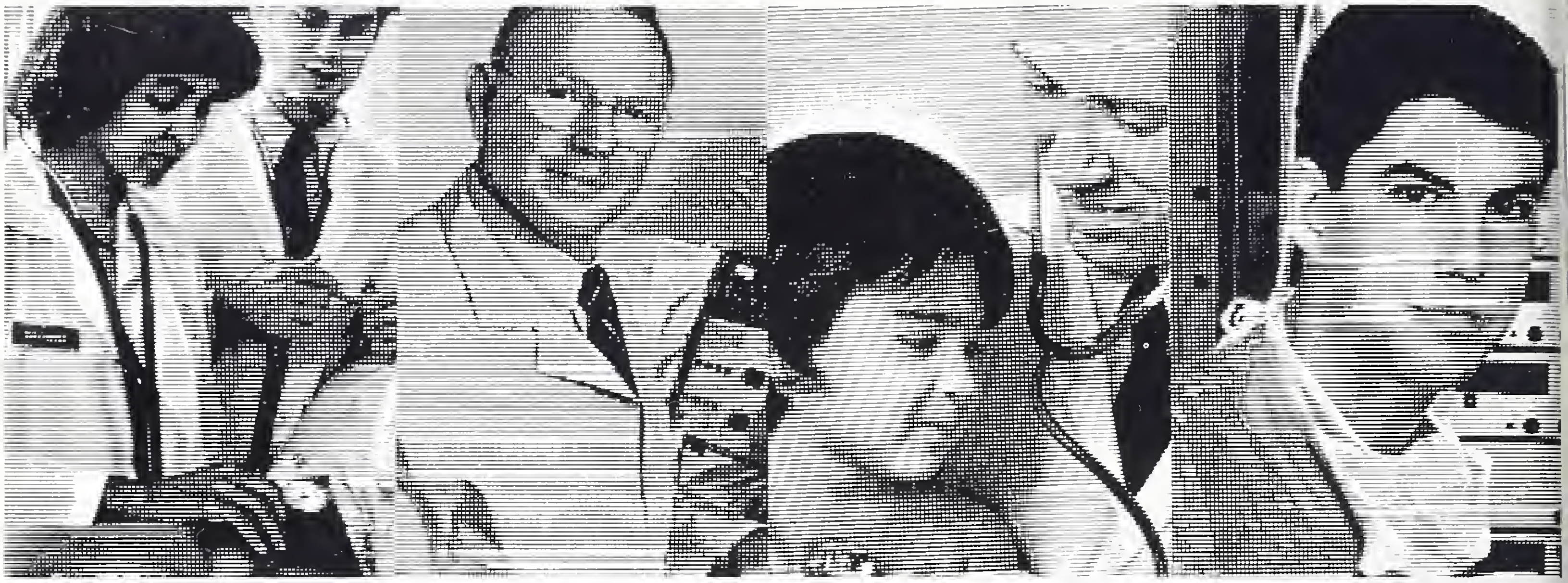

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> ***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.

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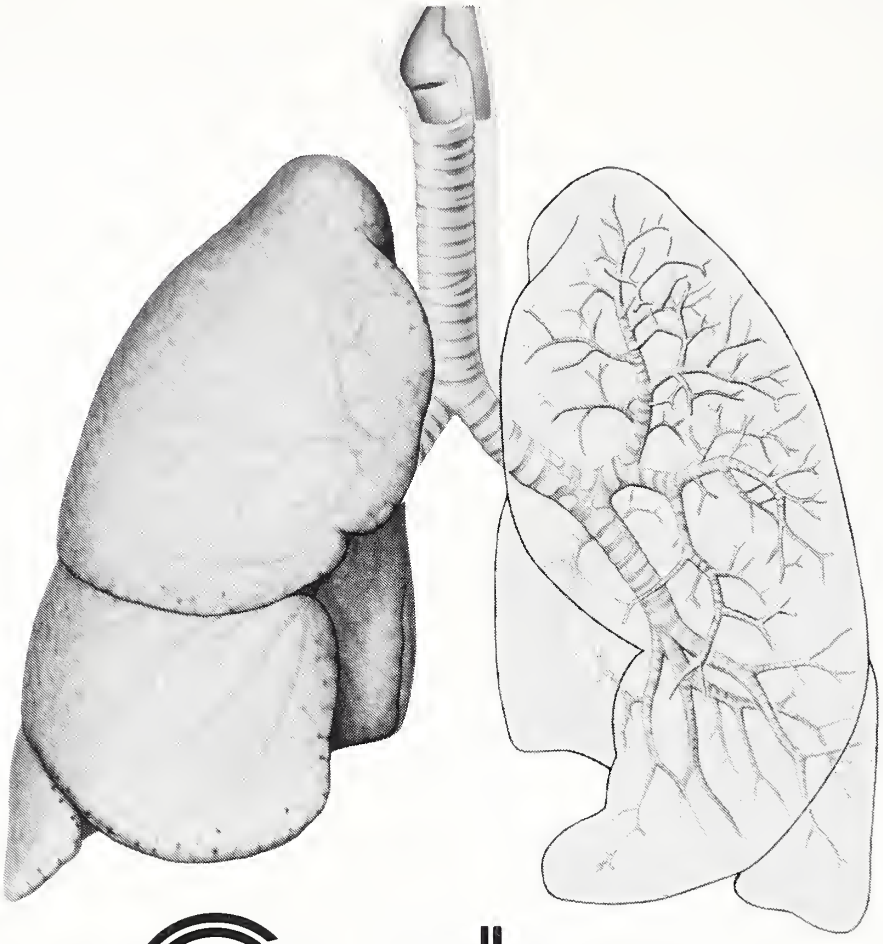

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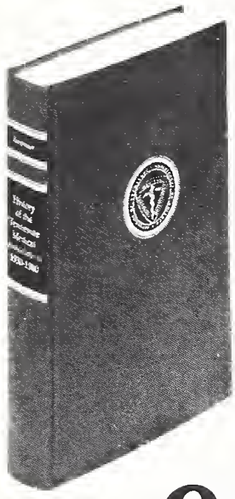

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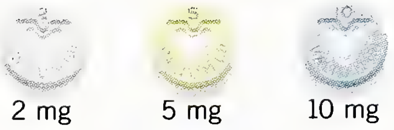

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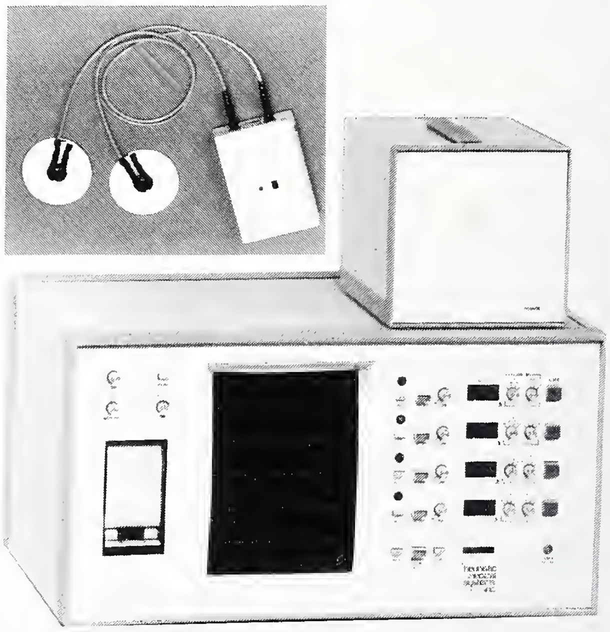

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Management of Diabetes Mellitus In the Home, Office and Hospital

ALAN L. GRABER, M.D.

Chronic management of diabetes is always a compromise between an ideal goal and what can be achieved for a particular patient. What can be achieved depends on a combination of biological, psychological, and social influences, in addition to the resources available to the clinician.[1]

The home is the most important site for chronic disease management, and therefore what the patient does daily at home is far more important than office or hospital encounters, which are like single frames on a movie film. Office appointments should be structured to update the home management process and to detect and prevent complications.[2] Since hospitalization is frequent in the diabetic population, it can also serve as an opportunity to reevaluate and reinforce home management techniques.[3]

When faced with a newly diagnosed patient with diabetes or a patient in very poor control, the first goal is to prevent acute metabolic emergencies such as ketoacidosis or the nonketotic hyperglycemic hyperosmolar syndrome. If this cannot be achieved in the home-office setting, hospitalization is required. Ketoacidosis and the hyperosmolar syndrome are hospital emergencies and their treatment is beyond the scope of this presentation. Providing insulin in adequate doses to lower serum glucose below the renal threshold usually relieves thirst, polyuria, and the acute symptoms of uncontrolled diabetes. The patient with new or uncontrolled diabetes must receive insulin and be instructed in insulin injection technique before instruction in diet, blood testing, and foot care. If the physician or his office personnel can do this, many hospitalizations can be prevented. The remainder of the patient education process can be pursued systematically, and is often more effective if spread out over several days or even weeks, rather than overwhelming a sick and frightened patient.

After resolving acute symptoms, the remainder of the diabetic patient's life is concerned with the avoidance of hypoglycemia and the sometimes abstract goal of preventing long-term complications. Adolescents and young adults have the most to gain from this process, but often have many other issues to distract them from focusing on possible health consequences 20 years in the future. On the other hand, once retinopathy or nephropathy are clinically evident, tight control of metabolic factors such as blood glucose has little effect, and most clinical efforts are focused on coping with the complications. Control of blood pressure has more influence on progres-

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From the Department of Medicine, St. Thomas Hospital, Nashville.

Presented as part of a course entitled "Outpatient Management of Chronic Medical Illness" at the Medical Learning Center, St. Thomas Hospital, Nashville, July 17-19, 1986.

Reprint requests to 2611 West End Ave., Suite 303, Nashville, TN 37203 (Dr. Graber).

sion of established nephropathy than control of blood glucose.

Some patients have inadequate glucose counterregulation, and are at increased risk of severe hypoglycemia with intensive insulin therapy.[4] These patients who can't respond to the usual cues for hypoglycemia may have repeated episodes of hypoglycemic coma if attempts are made to achieve euglycemia, and moderate hyperglycemia should be the goal of treatment.

In the absence of adequate defenses against hypoglycemia, optimal control of insulin-requiring diabetes should aim at near-normal control of blood glucose and normal levels of hemoglobin A_{1C}. This can be achieved in the majority of patients at least on a short-term basis, but has rarely been documented for prolonged periods.[5]

Following is an example of the ideal home management of a diabetic patient. An intelligent, mature young person has developed diabetes, has been started on insulin, and no longer has any acute symptoms of insulin deficiency; he now wants to actively devote all his efforts to minimizing the long-term effects of chronic hyperglycemia. He understands and acknowledges that neuropathy, microangiopathy, and premature vascular disease can be delayed or minimized by keeping his blood glucose normal, and is willing to do whatever is necessary to accomplish this goal over the next 40 years or so of his life.

The patient and clinician agree on setting a target of fasting serum glucose levels between 70 and 115 mg/dl. The level of 115 mg/dl is selected because it is the upper limit of normal in a nonpregnant adult.[6] Actually, microvascular progression has been demonstrated prospectively in populations with sustained fasting hyperglycemia above 140 mg/dl. In pregnancy the ideal target goal for fasting serum glucose would be 60 to 100 mg/dl, because this represents the range in nondiabetic pregnant women. After meals the glucose target range increases to 200 mg/dl in the nonpregnant individual, and to 150 mg/dl in the pregnant woman, but should return to the fasting level before the next meal. Random values above 200 mg/dl in the nonpregnant adult, or above 150 mg/dl during pregnancy, would indicate unacceptable control and would require immediate supplemental insulin. This type of metabolic control could be achieved in some stable type II diabetic patients who have considerable endogenous insulin reserve, but in our experience the average insulin-requiring type II diabetic patient has no better metabolic control than the average type I patient.

The achievement of this type of metabolic control in insulin-requiring diabetics, either type I or type II, requires frequent home capillary blood glucose monitoring.[7] This ideal patient agrees to perform this test eight times daily: before each meal, two hours after each meal, at bedtime, and about 2 AM, to assure that he does not develop asymptomatic undetected nocturnal hypoglycemia.

Our patient has unlimited financial resources, patience, family support, job security, and emotional stability. In addition, he eats his meals precisely as instructed at the same time each day, carefully counting both the number of diabetic exchanges and the grams of carbohydrates each time he eats. He exercises the same amount at the same time every day of each week, never gets sick, never gets pregnant, never goes on a trip, and never loses enthusiasm for precise control of his diabetes. In order to achieve the best control, he cheerfully purchases and wears an ambulatory, battery-powered insulin pump for chronic subcutaneous insulin infusion (CSII), despite its cost and inconvenience, and never gets a skin infection or has any discomfort at the needle insertion site. In addition, the needle never gets dislodged, even during exercise or sex, and the infusion tubing has never become obstructed.

Normal pancreatic insulin secretion has two phases, basal and meal-stimulated. The basal secretion, which remains fairly constant in the postabsorptive state, comprises about 40% to 60% of the total daily insulin secretion. The basal insulin requirement has been found to be 1.5 units/hr in this patient by a clinical test designed to determine basal insulin requirements.[8] The basal insulin infusion rate on the pump is therefore set at 1.5 units/hr, or 36 units/day, and the patient is given an algorithm to change the basal rate depending on the results of his blood glucose results at 2 AM and 6 AM. If there is an unexplained reproducible increase in blood glucose in the early-morning hours, the so-called dawn phenomenon, the insulin pump must be programmed to deliver a higher basal rate during these hours.[9] The patient administers his premeal insulin boluses at least 60 minutes before he eats, in order to allow time for absorption of subcutaneous insulin to coincide with absorption of ingested carbohydrate. Serum insulin levels reach a peak one to two hours after subcutaneous injection.[10] This explains the difficulty in minimizing postprandial blood glucose rise in patients who lack endogenous insulin secretion.[11]

Starting guidelines for premeal boluses average 1 unit/10 gm of carbohydrate ingested, but in reality different types of carbohydrate lead to greatly different degrees of postprandial hyperglycemia[12]; therefore the "glycemic index" of carbohydrates must be taken into account in determining the difference in premeal insulin dose for equivalent amounts of bread, potatoes, corn, or beans.

An example algorithm for this patient might be constructed as shown in Table 1.

If the patient does not use an insulin pump, combinations of intermediate or long-acting insulin with premeal regular insulin can be used. The principles of dosage and changes are similar to those described for CSII, but the results are less precise, primarily because of differences in rates of absorption from one patient to another. For example, the duration of regular insulin effect varies from four to eight hours, and the duration of NPH or Lente is even more difficult to predict. In addition, the time course of a dose of insulin varies depending upon the anatomical site of injection, with the rate of absorption highest in the abdomen, intermediate in the arms, and lowest in the legs. Muscular exercise in an extremity also increases the rate of insulin absorption. Given these limitations, algorithms may be prescribed for the twice-daily mixtures of intermediate-duration and regular insulin commonly used, but their effectiveness for ambulatory patients has never been established. Such algorithms, or guidelines for dosage changes, consist of changing a given insulin dose based on the preceding day's outcome of that dose, as follows:

Insulin Dose	Outcome Blood Glucose
Prebreakfast regular	Prelunch
Prebreakfast NPH or Lente	Presupper
Presupper regular	Bedtime
Presupper NPH or Lente	2 AM or prebreakfast

TABLE 1

ALGORITHM FOR INSULIN ADMINISTRATION IN A SAMPLE PATIENT

Basal Rate

FBS (mg/dl)	*Change Basal Rate*
<70	decrease 10% to 32 units/day
70-125	continue 36 units/day
>125	increase 10% to 40 units/day

Premeal Boluses

Premeal Glucose (mg/dl)	*Dose*
<70	0
70-125	1 unit/10 gm carbohydrate
125-200	1 unit/5 gm carbohydrate
>200	1 unit/5 gm + 3 unit supplement

Premeal boluses are changed by observing their effect on the outcome blood glucose obtained at least four hours later, or before the next meal, as follows:

Change Premeal Boluses, Based on Yesterday's Results

Prelunch Glucose (mg/dl)	*Change Prebreakfast Dose*
<70	decrease
70-125	no change
125-200	increase
>200	increase more

The ideal patient keeps accurate and reliable records of home blood glucose monitoring, but some patients have been shown to report values at home which are erroneously low either because of improper technique or deliberate falsification.[13] Therefore measurement of hemoglobin A_{1C} every three or four months is necessary to determine overall outcome of metabolic control. The hemoglobin A_{1C} test affords the best estimate of time-averaged blood glucose for the preceding three months. Misconceptions of both patient and physician regarding the effectiveness of control can be identified.[14]

Despite state-of-the-art outpatient medical supervision, including the provision of patient education, diet instruction, home blood glucose monitoring, and algorithms to change insulin dosage, the majority of diabetic patients remain in poor control. In a series of 233 patients we studied in the office, mean glycosylated hemoglobin (hemoglobin A_{1C}) and office blood glucose test results indicated average blood glucose values to vary between 200 and 300 mg/dl over a four-year period.[15]

The ideal patient makes an office appointment approximately every three months, at which time serum glucose and hemoglobin A_{1C} are determined. Metabolic control and insulin dosage are reviewed, but in truth a method should be established to accomplish this in a more timely fashion, for there is little interest in perusing results of home blood glucose tests from three months ago. The ideal patient removes his shoes and socks for the physician to inspect his feet at each office visit. Screening for complications, including examination of optic fundi, neurological status, urinalysis, and pulses, should be done systematically probably at least once a year in asymptomatic patients. Retinal photography is the best method for detecting asymptomatic proliferative retinopathy at a stage where intervention with laser treatment could prevent visual deterioration. A set of fundus photographs should be obtained early in the course of diabetes and repeated at intervals, though the optimal frequency from a cost/benefit viewpoint for this screening has not yet been determined. Ideally, blood pressure, serum lipids, weight, and other coronary risk factors will be kept normal.

The ideal patient will never require hospitalization, but about 10% of the diabetic population is hospitalized each year, usually for complications of diabetes or for reasons not directly associated with diabetes control. Vascular complications and lesions of the lower extremities account for the majority. When hospitalized, diabetic patients have longer hospital stays and a higher frequency of readmission than nondiabetic patients.

Though ketoacidosis, hyperosmolar coma, hypoglycemia, and other metabolic problems are relatively infrequent causes of hospitalization, provision for monitoring and controlling blood glucose during hospitalization is necessary for all diabetic patients, regardless of the reason for admission. In all cases, the physician should set a target range for blood glucose levels; we frequently set target ranges such as 80 to 200 mg/dl as acceptable during hospitalization. Either specially trained nurses or algorithms must be supplied to deal with hypoglycemia and hyperglycemia, both of which can be expected to occur in the hospital. Healing of infections or wounds should be enhanced and hospitalization should be shortened by these maneuvers.

Hospitalization strictly for metabolic control or patient education is becoming progressively infrequent. Though strict control of blood glucose can be achieved under hospital conditions and is sometimes necessary, e.g., for healing of infection, it cannot be expected to continue after discharge. When the patient is not too ill, education in self-care and diet should be provided in the hospital. In many communities hospitalization is the only opportunity for this. Ideally, this should be supplemented by outpatient programs when the patient is in his usual environment.

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A History of Cancer in U.S. Presidents

STEVEN HOLT STOKES, M.D.

Approximately one year ago America was rocked by Dr. Steven Rosenberg's sober statement, "The President has cancer." As we celebrate President Reagan's anniversary of continued good health following his successful surgery, it is of interest to reflect on other chief executives who have experienced this illness.

Ulysses S. Grant

It was June 1884, and former President Ulysses S. Grant's life was again in shambles. Not since his resignation from the Army in 1854, following allegations of alcoholism, had the future seemed so bleak. Three weeks previously the investment firm of "Grant and Ward" had collapsed. Ward, using Grant's political contacts and national reputation, had been successful in enticing potential investors, but in collusion with a banker, he used a portion of new investor money for dividends and embezzled the remainder. Inevitably the firm went bankrupt, leaving President Grant virtually penniless.

It was shortly thereafter that he noticed progressive pain in his throat. Upon the urging of his family a noted Philadelphia surgeon, J. M. DaCosta, was summoned. His initial evaluation, at the former President's home, identified a mass at the base of the tongue. DaCosta left a prescription and requested Grant contact his family physician, Dr. Fordyce Barker. DaCosta failed to notify Dr. Barker of his findings, possibly out of embarrassment for having examined another physician's patient without his consent, and Grant failed to mention that Dr. Barker was in Europe for the summer.[1]

Dr. DaCosta's prescription initially relieved the pain, but by October the discomfort had returned with increased intensity, and upon his wife's insistence he finally kept his appointment with Dr. Barker, who following an abbreviated examination immediately referred him to Dr. John Hancock Douglas, a noted otolaryngologist in New York.

Dr. Douglas, who had served on the U.S. Sanitary Commission during the war, had known the then "General Grant" well. On the morning of Oct. 22, 1884, four months after the appearance of his symptoms, Grant appeared in the office of Dr. Douglas.[2] The General explained his difficulty in swallowing and attributed his current illness to the recent shock of his recuperative powers (Grant-Ward failure) from which he had failed to recover. Douglas found the velum inflamed, of a "dark, deep congestive hue, a scaly squamous inflammation, strongly suggestive of a serious epithelial trouble." Upon completion of his examination, the only question Grant asked was, "Is it cancer?"

The question having been asked, says Dr. Douglas, "I realized that if he once found out that I had deceived him, I could never reinstate myself in his good opinion." I said, "General, the disease is serious, epithelial in character, and sometimes capable of being cured."[2]

Dr. Douglas consulted Dr. Shrady concerning the advisability of an operation. Shrady, who had previously attended Frederick III of Germany prior to his death from laryngeal carcinoma, recommended against surgery. The decision not to operate, which later generated intense controversy, "was manifested in sparing him unnecessary mutilation and allowed him to spend the rest of his days in comparative comfort. However, it meant suffering for him until the end."

At this point an individual appeared who, although not offering a chance of cure, at least offered him a purpose for living. Mark Twain had previously attempted, without success, to persuade Grant to write his memoirs. Twain, fresh from the triumph of Huckleberry Finn, accidently overheard a reporter at a lecture hall comment

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that General Grant had now decided to publish his memoirs. The reporter declared that in view of his financial distress, Grant was "gladdened" by the promise of $500 for each of three articles on disputed battles of the war, with the possibility of extending the effort into a book.

Next morning, Mark Twain called on General Grant to "smoke a cigar." He said to the General, "If, as I have heard, you are writing a book, I hope you make a good sum for yourself and family. As a friend, do you mind showing me the proposed contract?" Twain discovered that the publishers had offered a straight 10% royalty without advance payment and no guaranteed sales. Twain stormed that no man could be bound by such an inequitable contract. He immediately offered Grant $25,000 as an advance on expected royalties of the first volume and $25,000 for any subsequent volumes. The publishers, with whom Grant had contracted, were privately chagrined that someone else had "plowed with our heifer"[2] but did not attempt to enforce their contractual agreement. Grant again had a purpose for living. By writing his memoirs he could provide an estate for his wife and children.

Grant initially traveled twice a day by streetcar (he could not afford a private carriage) to Douglas' office, where the doctor would spray cocaine on the cancer, affording enough relief for Grant to swallow and continue writing. Later on, when Grant became too weak to travel, Dr. Douglas visited him at home, administering hypodermic injections of brandy and morphine, along with local applications of cocaine.

The newspapers criticized Grant's treatment, attacking Dr. Douglas and second guessing Dr. Shrady's original decision not to operate. Unsolicited "cures" were offered via the press, and of one newspaper Grant wrote, "It is an advertising medium for quack medicines prepared by ignorant people." This public support must have been a comfort to his physicians as they were forced to watch him deteriorate. Nonetheless, Dr. Douglas, by providing moderate relief of his pain, allowed Grant to work at a feverish pace through the winter and early spring.

On July 28, five days after mailing Volume II of his memoirs to the printer, Grant died quietly, his family and Dr. Douglas at his side. At the end he was at peace, knowing his wife and family would be solvent. Julia Grant's initial royalty check was for $200,000, with his memoirs ultimately generating half a million dollars. Like his Civil War triumphs, General Grant's final campaign had also been successful.

Grover Cleveland

On March 4, 1893, Grover Cleveland was inaugurated even as financial collapse of the U.S. Government loomed on the horizon, the result of the 1890 Sherman Act, which required the government to purchase yearly 54 million ounces of silver against which treasury notes were issued, all redeemable in gold. The world financial markets were on a gold standard alone, and foreign creditors required payment in gold for international transactions. This demand for gold resulted in rapid depletion of the U.S. Treasury's gold reserves. By inauguration day the Secretary was reduced to begging patriotic banks and bankers to let the government have their gold.[3] At his inauguration in March, President Cleveland declared he would exhaust all legal powers to prevent depreciation of the currency. Despite the impending crisis, Congress adjourned without enacting legislation to ease the financial situation.

On Tuesday, June 13, 1893, four months after assuming office, President Cleveland was visited socially by Major Robert O'Reilly, the White House physician.[4] While there, he was invited to examine a "rough place" on the roof of the President's mouth. He found an "ulcer as large as a quarter of a dollar, extending from the molar teeth to within one third of an inch of the middle line and encroaching slightly on the soft palate."[3] Dr. O'Reilly arranged for a long time friend and surgeon, Dr. Joseph Bryant, to examine Mr. Cleveland. Dr. Bryant quickly came to Washington and agreed with the diagnosis of malignancy. The President, after his examination, inquired: "What do you think it is, Doctor?" Dr. Bryant replied, "Were it in my mouth, I would have it removed at once."[3]

In discussing the plans for surgery, the President insisted on absolute secrecy, and the avoidance of even a hint of suspicion that anything of significance was pending. He decided that July 1 was the earliest possible date for surgery. Colonel LaMont, the Secretary of War, and a close personal friend, was the only one in government informed of the details.[3] To insure the best chance of secrecy, it was elected to perform the operation aboard the Oneida, the yacht of another friend, E. C. Benedict.

Dr. Bryant was a fortunate choice as the surgical consultant. Three years earlier he had addressed the Medical Society of New York on "A History of Two Hundred and Fifty Cases of Excisions of the Superior Maxilla," of which two of the cases were his.[4] In that series there was a 14% operative mortality, usually related to bleeding and infection. He was, therefore, well aware of the potential risk of the approaching surgery.

On the morning of June 30, Mr. Cleveland issued a call for a special session of Congress to convene on Aug. 7 for the purpose of resolving the financial crisis. That afternoon he departed the White House by train for New York harbor, arriving aboard the Oneida around midnight.[3,5]

After it had been decided that the surgery would be performed aboard ship, the Oneida had been the scene of intense activity. The saloon was prepared and disinfected. An operating table, necessary instruments, and drugs and medications were secretly brought aboard. A surgical team was quietly assembled. In addition to Dr. Bryant, the surgeon, additional members included Dr. E. G. Janeway, internist; Dr. John Erdmann, first surgical assistant; Dr. Ferdinand Hasbrouck, a dentist experienced in administering nitrous oxide, and Dr. O'Reilly.

Of major concern was the anesthetic, especially in a man of the President's physical condition. He was 56 years old and very obese, with a short neck; he was fatigued by four months of continuous conflict with Congress. It was decided that the initial teeth extraction would be performed under nitrous oxide, with ether reserved for the maxillary bone resection.

On the morning of July 1, the Oneida steamed up the East River at half speed while the operation proceeded. After the two upper bicuspid teeth were quickly extracted, an intraoral excision was made removing the lateral half of the hard palate, including the gingiva. It was found that the tumor had extended into the antrum superiorly, requiring excision of the floor of the orbit; by cautery and packing with gauze, bleeding was controlled and the surgery completed in one and a half hours. Throughout the procedure Dr. Janeway monitored the patient's general condition, Dr. Hasbrouck administered the nitrous oxide, Dr. O'Reilly the ether, and Dr. Bryant performed the surgery, assisted by Dr. Keen and Dr. Erdmann.[4]

Mr. Cleveland recovered rapidly and was out of bed on the second postoperative day. There was no evidence of infection, and he reached Gray Gables, the private presidential retreat, July 5, four days after surgery.

Unfortunately, two weeks later Dr. Bryant detected a persistent suspicious area on the inner margin of the wound and on July 17 the President again boarded the Oneida for additional excision and cautery of the abnormal area. As this was a less extensive procedure, he returned to Gray Gables the following day for continued recovery.

Rumors had begun to circulate that the President was seriously ill. Attorney General Olney, after several attempts, was finally allowed to see Mr. Cleveland. The President's mouth was packed with gauze and the first intelligible utterance was, "My God, Olney, they nearly killed me!"[5,6]

With gauze packing the surgical cavity, his speech, although labored, was intelligible, but with the gauze removed it was uninterpretable, resembling that caused by a severe cleft palate. To quiet newspaper speculation and reassure the public, Secretary Lamont held a press conference where it was maintained that the operation had been for the removal of a bad tooth. Dr. Bryant, despite considerable badgering by the press, refused to comment on the true nature of the operation. One enterprising reporter, in an effort to disprove the official story, even ferreted out the steward on the Oneida.

Dr. K. Gibson, a prosthodontist, established a dental laboratory at Gray Gables, where he fashioned a vulcanized rubber prosthesis; it was so skillfully crafted that once it was in place the President's speech was excellent, without alteration in its quality.[3]

On Aug. 7, the President appeared before Congress asking for repeal of the Sherman Silver Act. Shortly thereafter, the *Philadelphia Press* published a fairly accurate account of the President's cancer surgery. Dr. Hasbrouck had carelessly confided to a friend the details of the operation, who then related them to a reporter. Dr. Bryant was so upset by the breach of secrecy that he never again spoke to Dr. Hasbrouck and sent him a one dollar fee by a messenger for his services.[5]

The White House denied the story and labeled it a spurious campaign by the Silver Interest to discredit the President's efforts for repeal of the Sherman Act. In view of President Cleveland's having opened the special session of Congress with no evidence of a scar and in perfect voice, the newspaper account was discredited.

President Cleveland had no further difficulty with his cancer. He survived for an additional 15 years, ultimately dying of heart failure. It was not until Dr. W. Keen published his account in the 1917 *Saturday Evening Post* that the public was made aware of the seriousness of the President's operation, which contrasts sharply with President Reagan's surgery. Here the most intimate details were openly discussed, leaving the White House staff fuming over the "tasteless" drawings of the President's bowels that appeared in some newspapers.[7]

Ronald Reagan

During a routine examination in May 1984, a hyperplastic polyp was removed from the President's lower colon.[8] No further examinations were performed until March 1985, when an additional small hyperplastic polyp was detected. However, the more ominous findings of occult intestinal bleeding prompted the recommendation for a more thorough colonoscopic evaluation of the entire colon. Due to the demands of office, not until July was President Reagan able to enter Bethesda Naval Hospital for this evaluation.

The colonoscopy had been expected to be a routine 30 to 45 minute procedure to search for and remove any additional polyps. To the physician's surprise, a large villous adenoma, a known premalignant lesion, was encountered near the ileocecal junction.

President Reagan, on being informed of the findings, elected to proceed with surgical removal the following day. Despite an initial biopsy showing no evidence of tumor, the hemicolectomy specimen demonstrated invasive carcinoma. Fortunately, there was no evidence of nodal metastasis, and the tumor was confined to the wall of the colon, both of which findings predict an excellent chance of cure.[9]

Summary

In reviewing the details of how each president responsed to cancer, there is one central theme. All demonstrated remarkable courage and overriding concern for the welfare of others. Grant's goal was to survive long enough to complete his memoirs providing an inheritance for his wife and family. Cleveland would consent to surgery only once he was assured secrecy could be maintained so as not to jeopardize the economic stability of the country. President Reagan immediately resumed the responsibilities of his office despite major surgery.

The diagnosis of cancer has always elicited fear. Each president refused to allow his fear to overwhelm him. Instead, in each case the individual proceeded with the best therapy available. Each maintained control of his life, thereby setting an example for all of us with a similar diagnosis to emulate.

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Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
			1	2	3	4
5	6	7	8 TMA 152ND ANNUAL MEETING Hyatt Regency Hotel—Knoxville	9	10	11
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Sickle Cell Disease and Sudden Death

IFEANYI O. OBIANYO, M.D. and ROBERT E. HARDY, M.D.

Sickle cell trait, the heterozygous state (Hb SA), is present in 8% of American blacks and in as many as 30% of some African populations.[1] Initially described by Diggs[2] as usually a harmless carrier state or condition rather than a disease, it has been known to cause spontaneous hematuria,[3] and renal[4] and splenic[5] infarctions. Following the review of deaths attributed to sickle cell trait in 1964 by Schenk,[6] a number of cases involving sudden death from sickle cell heterozygous states have been reported.[7,8]

Sickle hemoglobin, the homozygous state (Hb SS), so called because of the sickle shape it imparts to deoxygenated red cells, is responsible for a wide spectrum of disorders that vary as to degree of anemia, frequency of crises, extent of organ damage, and duration of survival. This disease has been recognized as a lifelong debilitating disease, serious and frequently lethal, culminating in multiple hospital admissions. Rheologic abnormalities characteristic of this anemia have resulted in acute episodes and resultant chronicity leading to organ damage. Recurrent vaso-occlusive crises, which are a major feature of this disease, have resulted in renal, hepatic, skeletal, and neurologic dysfunction. Sudden death in sickle cell disease patients is increasing, though only a few cases have been reported. We describe here two cases of sudden death in patients with known sickle cell anemia.

Case Reports

Case 1. A 33-year-old black man was admitted to Hubbard Hospital in October 1984 because of two weeks of dyspnea at rest, pleuritic chest pain, leg edema, dry hacking cough, and fever.

In 1953, when he was 2 years old, he was diagnosed as having sickle cell anemia, Hb SS. He had been having an average of three crises per year, mostly triggered by infections. He had right lower lobe pneumonia in September 1981, bilateral pneumonia in February 1983, and cryptococcal pneumonia in October 1984. He had a cholecystectomy for cholelithiasis and cholecystitis in May 1983, and congestive cardiopathy with an ejection fraction of 30% in October 1983. He had smoked one pack of cigarettes every day for 15 years and worked as a school bus driver.

At the time of his last admission to the hospital, he had signs of congestive heart failure with a blood pressure of 120/90 mm Hg, pulse 80/min, and regular respiration at 22/min. Neck veins were distended, with an estimated jugulo-venous pressure of 12 cm H_2O. He had bilateral inspiratory and expiratory wheezes with rales and rhonchi more marked in the right lung fields. Cardiac examination revealed a normal sinus rhythm, regular rate, and an S_3 gallop. His abdomen was distended, with the liver percussed at 18 cm in the anterior axillary line without fluid waves.

He had marked scleral icterus, pale conjunctivae, and right anisocoria. Leukocyte count was 15,000/cu mm, with 79% segmented neutrophils, 2% bands, 15% lymphocytes, and 4% monocytes; hematocrit was 26.7% with a reticulocyte count of 14.7%. Chest x-ray revealed cardiomegaly, with left atrial and biventricular enlargement and consolidations of the right lung. Fiberoptic bronchoscopy revealed chronic bronchitis, and bronchial secretions contained *Cryptococcus neoformans*. Cerebrospinal fluid analysis and blood cultures were negative.

This patient was treated with amphotericin B for his pneumonia and for heart failure; both improved. He was subsequently discharged but was readmitted two weeks later for treatment of local infection at the catheter site. After catheter replacement he returned home on continued antifungal therapy which resulted in further improvement.

On two follow-up visits, he appeared to be asymptomatic and doing well without any further complications, but on Jan. 30, he suddenly had a cardiac arrest at home and died. No autopsy was performed.

Case 2. A 35-year-old black man was admitted to Hubbard Hospital in March 1985 for iron overload and for right hip and back pain of 10 years' duration which progressively worsened and subsequently culminated in aseptic necrosis of the femoral heads.

In 1957, when he was 3 years old, he was diagnosed as having sickle cell anemia, Hb SS. He had an average of four crises per year, usually associated with infection. He had two episodes of pneumococcal pneumonia and several superficial skin ulcers of his right ankle. He also developed salmonella osteomyelitis of the tibial tuberosity six weeks prior to the March admission.

At time of last admission, he was unable to walk due to severe right hip pain. He had a temperature of 98°F, regular pulse of 84/min, respiration of 18/min, and a blood pressure of 140/80 mm Hg. His conjunctivae were pink, and both pupils were equally reactive to light. Lungs were clear with good breath sounds, and heart showed normal sinus rhythm and a grade 2/6 systolic ejection murmur. The medial aspect of the left knee drained a serous exudate but there was no edema. The midlumbar spine was tender, and the right hip was markedly tender with decreased range of motion.

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Repeated cultures of left knee exudate were negative. Hematocrit was 25.9%, with a reticulocyte count of 6.6%. Serum iron was 63 μg/dl, TIBC 223 μg/dl, saturation 31%, and ferritin 4,510 ng/ml. Chest x-ray revealed cardiomegaly, platelike atelectasis in the right base, and fishmouth vertebrae in the dorsal spine. X-ray of lumbar spine revealed some fish-mouthing and osteophytic changes; x-ray of right and left hips revealed aseptic necrosis of the femoral heads, with coarsening and trabeculi on the right.

A bone scan revealed abnormal uptake in the left frontal sinus consistent with sinusitis or hematoma, and osteomyelitis in the lower lumbar vertebrae and left proximal tibia. The platelet count was 680,000/cu mm and the erythrocyte sedimentation rate was 39 mm/hr. A bone marrow aspiration revealed erythroid hyperplasia, increased thrombopoiesis, and increased iron stores.

He underwent saucerization of the left knee ulcer and chelation therapy for iron overload. Right total hip replacement was planned but not done because of his active bone infection. He improved markedly and was subsequently discharged, and on follow-up visits he did well and showed signs of decreased wound drainage.

On May 12, he developed sudden onset of excruciating chest pain and was rushed to a nearby hospital, where he died. No autopsy was performed.

Discussion

These two cases have some striking features: they have sickle cell anemia, with subsequent development of cardiac enlargement, and one of them congestive heart failure. Both were asymptomatic 24 hours prior to death. It has been demonstrated in some sickle cell patients that cardiac enlargement predisposes to pulmonary embolism arising from mural thrombi, and it is believed that patients with sickle cell states have an increased susceptibility to pulmonary embolism with infarction. A review of pulmonary complications in patients with sickle cell disease reveals repeated episodes of in-situ pulmonary thrombosis with subsequent cor pulmonale,[10] although convincing hemodynamic or autopsy data are few. Moser and Shea[11] described a possible relationship between cor pulmonale due to pulmonary thromboembolism and sickle cell anemia; they found that patients with large amounts of hemoglobin S were prone to develop pulmonary artery thrombi under conditions of reduced alveolar oxygen tension.

Robler and Fleischer[12] suggested alcohol as a precipitating agent of pulmonary thromboembolism in patients with susceptible sickle cell trait.

Jones et al[13] described 12 deaths attributed to sickle cell hemoglobinopathy of which a majority was due to thrombotic episodes or concomitant illness.

Myocardial infarction, a known cause of sudden death in older, non-sickle cell patients, is not a significant cause of death in sickle cell patients. Gerry et al,[14] who looked at the clinicopathology of cardiac dysfunction in 52 patients with sickle cell anemia, found renal failure and infection the most common causes of death in these patients. Right and left ventricular hypertrophy and dilation were the most abnormal pathologic findings. No evidence of recent or remote myocardial infarction, coronary thrombosis, or arteritis were noted. Barrett[10] reviewed the electrocardiograms of 108 patients and found only three with patterns consistent with myocardial infarction.

Conclusion

An extensive review of the literature has denoted a significant number of individuals dying suddenly with sickle cell trait and homozygous sickle cell anemia. Most autopsies in these patients have revealed pulmonary infarction due to thromboembolism, with pulmonary hypertension as the most probable culprit; decreased pulmonary oxygen tension is believed to be the triggering event.

This paper describes two cases of known sickle cell anemia with resultant sudden death. Although no autopsies were performed, it can be reasonably inferred that pulmonary thromboembolism was the main cause of death. Since a number of cases of sudden death in patients with sickle cell trait have been reported, one would expect an even higher incidence of sudden death from this cause in patients with sickle cell anemia. Predisposing conditions leading to sudden death in subgroups of sickle cell anemia patients at risk should be identified for active intervention, treatment, and preventive measures.

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Special Item

Should Your Children Seek To Become Physicians?

LEIF C. BECK, J.D., CPBC

In my constant discussions with privately practicing physicians, I so often hear a terribly unsettling statement: "I've enjoyed medical practice until recently, but I certainly hope my son (or daughter) won't go into medicine." While a certain level of frustration as to the changing scene is understandable, I cannot agree with the thought process behind that comment.

It puts directly before us a basic question whether the medical profession has really declined at all and, if so, whether the decline is so severe that the profession is now undesirable to existing physicians and their children. On my broad-based involvements with thousands of doctors under a wide variety of personal and professional concerns, I do not equate the changing scene with decline. And if one terms the changes (both already occurred and rapidly coming on) as "decline," they still leave medicine as a highly desirable future for bright, well-motivated young people.

The "Golden Era"

Consider how presently successful physicians came to our present times. Practicing in the 1960s and 1970s, they participated in the "golden era" of private practice. There was an explosive growth of new diagnostic capabilities, curative therapies, and dramatic procedures which made medical practice intellectually demanding but exhilarating. And an overwhelming supply of patients presented to each good physician—as well as too many not-so-good doctors. As a result, physicians succeeded both professionally and financially, often in spite of their own clinical or business capacities.

Physicians truly acted as the captains of health care. They were almost god-like in both the hospital and the office; they could bask in that role. There was tremendous ego satisfaction from their role and their crowded waiting rooms. The overwhelming demands on their time and energy merely fed the ego satisfaction even more.

These factors were naturally accompanied by disproportionately high incomes compared to other vocations. Though physicians pointed to their incredibly long hours of training and work to show that they were paid less than the average plumber, plumbers would have been delighted at the opportunity for such high total earnings.

The so-called golden era was, however, somewhat unreal and not wholly desirable. Patients had little alternative but to wait untold weeks for appointments and hours to be seen. They had to trust in the diagnosis and advice given by a selected physician whether he (or occasionally, she) was highly regarded by his peers or thought of as a poor clinician.

Personally, that era's physicians were so driven by the system and its unavoidable demands that their lives were too often totally one-dimensional. I considered it routine to hear complaints of an inability to participate in other worthwhile activities or even to have any other interests to occupy retirement years. Divorces, stale marriages, and lack of parental involvement were common problems. In effect, professional practice activities became these physicians' entire lives.

Time Moves On

Other parts of society rather naturally reacted to the golden era's circumstances. Consumerism developed in all aspects of our economy interfering with the public's respect for physicians as authority figures. Even if one's doctor technically knew what should be best for a patient, that individual began insisting on the right to decide for himself or herself.

Malpractice actions have, of course, skyrock-

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eted in both number and physician frustration. As over-busy doctors lost the time and energy for one-on-one involvement with patients, augmented by technicological gadgetry implying infallible results, the trend has been somewhat understandable.

Employers, insurance companies and government—the third parties which paid the most medical bills—began to rebel against the costs involved. Physicians' fees were only a moderate part of the health care cost picture, but the total amounts either paid to or directed by doctors were growing out of synch with society's other perceived essentials.

Other businesses saw the health care industry's profit potential and resolved to compete for slices of it. When hundreds of billions of dollars are involved, much within the physicians' control, the inflow—even the domination—by "big business" could hardly be avoided. And while I do not necessarily espouse it, there is nothing logically improper in a business corporation's directing a medical practice staffed by physicians doing what they are trained to do—deliver technical services.

Despite these reactions within the system, more and more young people clamored for medical careers. An M.D. degree had for so long been synonymous with success in life—high income, intellectual challenge and human service—that the increasing doctor supply is hardly surprising.

The Turbulent Eighties

In effect, the times changed—as they always do. Every other industry in our society is undergoing wrenching change, some far more traumatic than for physicians, so medicine cannot expect otherwise. Medical practice's features today are different from those of the golden era, which are vastly changed from those of the 1930s and 1940s, and so on. "The more things change, the more they remain the same."

So here we are in the "turbulent eighties." Medicine, just as the other professions, is in considerable turmoil as the various pincers of supply, demand, technology and activism attack traditional practice styles. These factors will not stop, indeed they are just warming up. Our society is too dynamic for any other scenario.

The New Generation

Young people enter this perspective from a different view of life. I sympathize with medical students, residents and fellows who entered medicine in reliance on the golden era, but a considerable segment of this generation suggests another set of priorities. Their interests seem to emphasize completeness of life, commitment to quality and service, aversion to personal/financial risk, and satisfaction with only moderately high incomes.

A page one article in the *Wall Street Journal* on Jan. 13, 1986, may have summarized some of these features by its headlines and subheadings: "More young doctors shun private practice work as employees; HMOs and big groups free them from bad hours, hassles of small business; home for dinner on time." Among the article's major quotations were these: "We want to be good parents and spouses as well. . . . Workaholics make good doctors, lousy fathers." Nothing in the article suggested—nor do we detect—any lesser commitment to concern for the patient or compulsion to provide high quality service.

And who are we to say their sense of priorities is wrong? A vastly increased proportion of these young physicians are women. They have become a valued resource in medicine, but they also possess natural—indeed biologically and socially necessary—instincts for family. And in tandem with the women's movement and two-income families, more physicians seem oriented to moderately paying positions of intellectual and/or human service orientation than in the past. I believe these people are a real prediction of the next generation of doctors.

The other segment of the current generation, including doctors who have entered practice in just the last few years, demonstrate the golden era's priorities in a shifted scene. They entered medical school in the background of high incomes and hearty self-determination. Some will hook on, or have hooked on, with excellent practices likely to ride on in the old style for quite a while; others of them have not and will not.

Young doctors committed to traditional medical practice style have the greatest stake in maintaining those features for the next 20 and 30 years. With so many pressures going the opposite way, it is not surprising that these doctors must engage in such concerns as entrepreneurialism and marketing to attempt to retain their practices' dominant roles.

Your Children

But the next generation—the children whose physician-parents say they would not encourage into medicine—will start with a clean slate. They will know that medical practice will be no financial gravy-train, but I believe they can still expect comfortable incomes. In fact, I predict that physicians' incomes will continue rather indefinitely to be the highest among generally compared professions and vocations even though they will be proportionately lower than in the past.

These young people can enter medicine because of their commitment either to the intellectual challenge of solving the human body's riddles or to the opportunity of serving other people's needs, or both. They should expect a greater likelihood that those commitments will be accomplished without the disturbances of overwhelming practice demands that changed their doctor-parents' lives. And they will hope to do so within more balanced lives in which family priorities and other valuable concerns and interests have a chance to blossom.

For those potential physicians who have the characteristics, I expect ample opportunities to be valued as medical practice entrepreneurs and executives. Health care will always require good physician-leaders, and leadership traits are becoming increasingly valuable these days. There *will* be private medical practices—a great many of them—and they will offer opportunities for clinically excellent doctors and somewhat separately for effective leaders. Progression from one category to the other will be not unlike an engineer's promotion from his or her peer ranks into a large commercial corporation's executive track.

That's not a bad scenario for your children even though it is vastly different from yours. The very special values of a medical career will not change, and they may well be allowed to shine. Perhaps the recent words of C. Everett Koop, M.D. (*Pa Med* 88:31, Dec 1985), a vastly respected medical practitioner throughout the "golden era" and now Surgeon General of the United States, state the case so well:

". . . I envy those starting a career in medicine. I regret some things that are going on—the lack of good doctor-patient relationships, the preoccupation with money and power, the growth of regulation, and so on. But I also know that medicine still offers the individual one of the most satisfying ways to make a life that I have ever encountered."

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Medical Grand Rounds

Macrocytic Anemia

CHARLES E. KOSSMANN, M.D., Editor

BRENT WELCH, M.D.
(Resident Physician)

A 41-year-old black woman came to the MED emergency room in July 1985 with an eight-month history of gradually increasing weakness, easy fatigability, lethargy, and malaise. She further described approximately two months of intermittent swelling and soreness of her tongue, and one month of rapidly progressive worsening of weakness to the point at which with minimal exertion, such as dressing in the morning, she had shortness of breath and a dull substernal, nonradiating chest pain, not associated with sweating, nausea, or vomiting. This exertional pain was relieved by rest within one to two minutes.

She denied any past medical history or chronic medical problems except that in the past at another hospital she had been diagnosed as having anemia, but she was unable to characterize the anemia or any treatment received for it. She denied taking any prescription or over-the-counter medicines. There was no recent weight loss, fever, chills, night sweats, nausea, vomiting, change in appetite, melena, hematochezia, or change in character of stools. Easy bleeding or bruising had not been noted and arthralgias or rashes were denied. She had no neurologic complaints, including paresthesias, ataxia, change in memory, increase in irritability, or change in the senses of taste, smell or vision.

She had smoked one pack of cigarettes per day for approximately 20 years but stopped approximately two weeks before admission because of shortness of breath. She denied any alcohol use or toxin exposures. She worked as a cook in a local school cafeteria and ate a normal diet, including meat and vegetables.

Her oral temperature was 99.0° F, ventilatory rate 20/min, heart rate 104/min, blood pressure 100/60 mm Hg without an orthostatic fall. She was a pale, thin, black woman in no acute distress, alert and oriented. The sclerae were anicteric, the ocular fundi unremarkable. Mucous membranes were pale, without petechiae or other lesions. There was slight erythema of the tongue, with mild papillary atrophy. Except for tachycardia and a systolic crescendo-decrescendo murmur at the lower left sternal border which radiated into the carotids, the heart and lungs were negative. The liver was enlarged 8 cm below the costal margin and nontender. On rectal examination the stool was guaiac negative. Cranial nerves II to XII were intact, as were all motor and sensory functions including proprioception, vibration, light touch and pain.

The hematocrit was 8.9%, the hemoglobin 3.1 gm/dl. Red blood cell indices were: mean corpuscular volume (MCV) 107 fl (normal 87±5 fl)*; mean corpuscular hemoglobin (MCH) 38 pg (normal 29±2 pg); mean corpuscular hemoglobin concentration (MCHC) 35% (normal 34±2%), and red cell distribution width (RDW) 25% (normal < 15.0%). The white blood cell (WBC) count was 2,900/cu mm; 17% were segmented, 81% were lymphocytes, 1% monocytes, and 1% eosinophils. The platelet count was 37,000/cu mm and the erythrocyte sedimentation rate was 74 mm/hr. The blood smear showed macrocytic red cells with mild hypochromia, marked anisocytosis, and numerous bizarre forms, including ovalocytes, target cells, and fragments. There were many hypersegmented polymorphonuclear neutrophils (Fig. 1); the lymphocytes and platelets were normal in appearance. The urinalysis and the serum sodium, potassium, chloride, bicarbonate, prothrombin time, and partial thromboplastin time were all within normal limits. The reticulocyte count was 3.3%. The chest roentgenogram was unremarkable. The electrocardiogram showed a sinus tachycardia but was otherwise within normal limits.

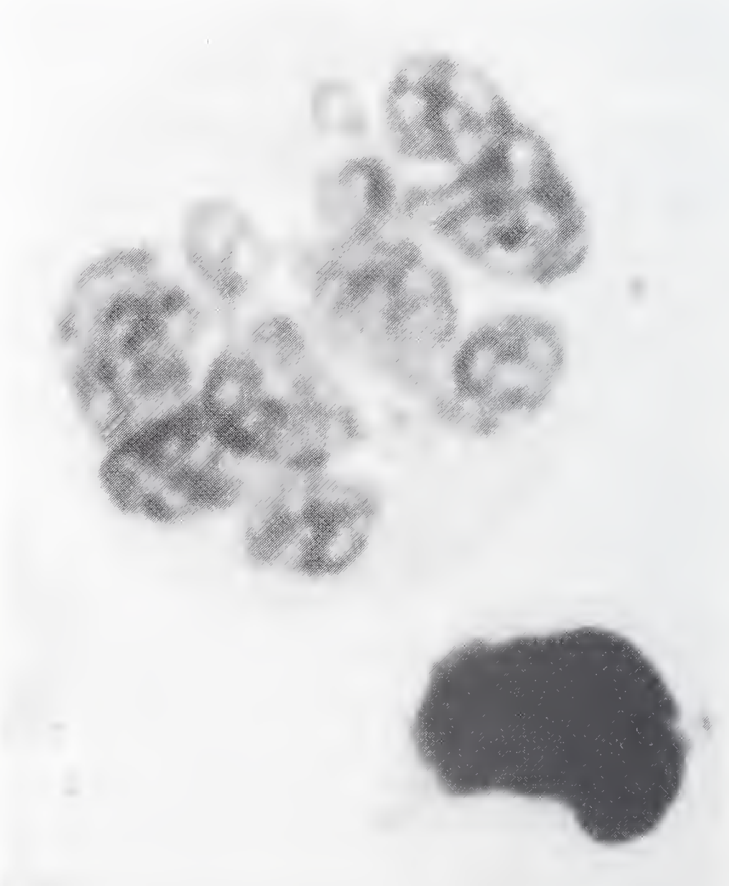


Figure 1. Hyperlobulated neutrophil on peripheral blood smear from a case of pernicious anemia.

From the Department of Medicine, University of Tennessee College of Medicine, 951 Court Ave, Memphis, TN 38163.

Presented Aug. 21, 1985.

*fl = femtoliter (1 quadrillionth of a liter—1 × 10^{-15}L)

The patient was hospitalized for further evaluation of pancytopenia, placed in protective isolation initially because of neutropenia, and blood drawn for appropriate studies. These revealed a total serum bilirubin of 1.6 mg/dl, with 1.3 mg of this being the indirect form. The aspartate aminotransferase (SGOT) and the alanine aminotransferase (SGPT) were slightly elevated at 122 and 79 U/L respectively. The lactic dehydrogenase (LDH) was markedly elevated at 2,019 U/L. The fluorescent antinuclear antibody (FANA) was negative. Response to intracutaneous PPD was negative, with positive mumps control on skin testing. Examination of the bone marrow revealed erythroid hyperplasia with typical megaloblastic erythropoiesis. There were numerous mitotic figures, giant band forms, and metamyelocytes. Histiocytic iron was found to be decreased, with a slightly increased sideroblastic iron. Serum iron and ferritin were normal, and the total iron binding capacity (TIBC) slightly reduced.

The patient was transfused slowly with four units of packed red blood cells with prompt resolution of the chest pain and shortness of breath on effort. Therapy with IM injections of B_{12}, 1,000 μg/day and folate 2 mg/day were begun. The initial low serum level of B_{12} was less than 30 pg/ml; folate level was normal at 16 ng/ml. Studies made approximately six days after the initiation of therapy with B_{12} and folate showed reticulocytes increased to 10.2% of red cells, hematocrit to 32.2%, white blood cells to 4,400/cu mm, and platelets to 90,000/cu mm. Serum assay for anti-intrinsic factor antibody was positive.

The patient was taught self-administration of IM injections of vitamin B_{12} and was discharged on the 10th hospital day with instructions to take daily injections of 200 mg of B_{12} for four weeks, then 100 μg each month with regular follow-up in the medical clinic.

Final Diagnosis: Pernicious anemia.

DR. LUTHER L. BURKETT
(Professor of Medicine, Hematology/Oncology)

I should like to point out some pertinent features of this case. First, she was a 41-year-old black woman. A number of years ago it was the general impression that pernicious anemia (PA) was predominantly a disease of elderly whites and rare in blacks, which has not proven to be true. Studies of patients hospitalized for PA have shown that the incidence in blacks, when adjusted for the ratio of white to black admissions, approaches the incidence in white patients. An earlier onset of the disease, particularly in black women, has also been observed. In one study of black patients with PA,[1] the mean age of onset was 53 years, compared to a mean onset of about 60 years in whites. Thirty percent of the black patients were under the age of 40 years, compared to only 11% of the white patients. Eleven percent of the black patients were under 30 years of age, which is quite uncommon in young whites.

The patient complained of a sore tongue, though she did not have the slick, glazed tongue sometimes seen in PA, but the papillary atrophy was recognizable because of the contrasting prominent circumvallate papillae. She herself had noted what she referred to as large red spots on the back of her tongue.

Although our patient did not have a palpable spleen, physicians often are surprised to find an enlarged spleen in PA, even though splenomegaly is seen in about 12% of patients at presentation. It is somewhat unusual that she had no neurologic manifestations at all; nowadays we do not often encounter the extreme neurologic deficits that were common a number of years ago, but most patients do have some neurologic problem such as subjective paresthesias, stocking-glove sensory loss, and early loss of vibratory perception. It has been suggested that early proprioceptive loss can best be detected using a tuning fork vibrating at 256 vibrations/sec^2 rather than the usual 128 Hz.

The patient had a profound pancytopenia, the usual hematologic abnormality in PA, although the neutropenia was a bit more severe than usually seen. It is to be noted that her MCV was not very low in spite of the profound anemia, which we will discuss later. She had an indirect hyperbilirubinemia, which is usual, due to both intramedullary destruction of red cell precursors and intravascular hemolysis. In the more severe anemias one finds spherocytes, fragmented red cells, a low serum haptoglobin, and a shortened red cell survival time. The LDH is greatly increased, sometimes to levels of 10,000 U/L or more. The

TABLE 1*

INCREASED MCV (> 100 fl) IN THE ABSENCE OF COBALAMIN OR FOLATE DEFICIENCY

- Common causes
  - Alcoholism
  - Liver disease
  - Cancer chemotherapy
  - Bone marrow malignancies:
    - Myeloma, leukemias, lymphomas, metastatic carcinoma
  - No cause apparent
  - Reticulocytosis
- Relatively uncommon causes
  - Hypothyroidism
  - Bone marrow disorders:
    - Aplastic anemia, pure red cell aplasia, refractory anemia, sideroblastic anemia, myelofibrosis
  - Hereditary hydrocytosis
- Laboratory artefacts
  - Cold agglutinins
  - Marked hyperglycemia
  - Marked leukocytosis

*From Lindenbaum[3] with permission.

TABLE 2*

CONDITIONS ASSOCIATED WITH MACROCYTOSIS, BY DEGREE OF ANEMIA†

	None	Mild	Moderate to Severe	All Degrees
Folate deficiency	15	13	3	31
Vitamin B_{12} deficiency	3	7	3	13
Both	1	3	2	6
Liver disease‡	6	7	2	15
Alcoholism only	8	3	—	11
Other conditions	7	8	9	24
All conditions	40	41	19	100

* From McPhedran et al[4] with permission.

† Degrees of anemia (hemoglobin level) for males: none, 14+ gm/dl; mild, 10 to 13.9 gm/dl; and for females: none, 12+ gm/dl; mild, 9 to 11.9 gm/dl.

‡ In the absence of folate or vitamin B_{12} deficiency.

increase is in fractions 1 and 2, with a relatively greater increase in fraction 1, in contrast to what is found in hemolytic anemias.

Macrocytosis

A review article on the laboratory diagnosis of megaloblastic anemias in 1983 by John Lindenbaum[3] begins, "What was once relatively straightforward seems now to have become more confusing," and goes on to cite the reasons for this confusion in diagnosis. First, the development of automated cell counters has allowed us to recognize lesser degrees of macrocytosis than was possible by earlier methods of counting or by examining the blood smear. Consequently, macrocytosis is appreciated more frequently, and many more causes have been recognized. Secondly, there has been the development of what Lindenbaum refers to as easy-to-do radioisotope dilution assays for serum B_{12} and folate, which generally have been accepted to reflect the state of vitamin metabolism accurately and reliably when often they do not. Further discussion will address some of the problems in diagnosis which even in standard textbooks of hematology may be presented inaccurately in substance, or incompletely in detail.

Table 1 lists causes of macrocytosis other than B_{12} or folate deficiency[3]; probably the most common cause of a slight or moderate increase in the MCV is alcoholism, with or without hepatic disease. Other common causes include cancer chemotherapy, which interferes with DNA synthesis and reticulocytosis, since reticulocytes are macrocytic. Often a cause for the increased MCV is not apparent. Occasionally it may be due to chronic obstructive pulmonary disease for reasons that are not clear. Less common causes are nevertheless important, since the finding may provide a clue to diagnosis. Hypothyroidism in the absence of B_{12} or folate deficiency may be associated with a macrocytic anemia. Large red cells are frequently seen with stem cell disorders, particularly aplastic and refractory anemias.

Occasionally an increased MCV is laboratory artefact. Cold agglutinins cause red cells to clump at room temperature, and cells going through the aperture of the counter in doublets or triplets are interpreted as single large cells. When the blood sugar is over 600 mg/dl the high intracellular osmolarity causes fluid to be drawn into the cell when placed in isotonic diluent, and a spurious macrocytosis results if the count is done before equilibration. The larger unlysed white cells are included in the particle count for red cells such that a marked leukocytosis, particularly chronic lymphocytic leukemia, produces a spurious macrocytosis.

McPhedran et al[4] evaluated 100 consecutive patients in whom there was an increase in the MCV of 115 fl or greater (Table 2). Approximately half of the patients had either B_{12} or folate deficiency or both. In the remainder the common causes were hepatic disease and alcoholism. Others were patients receiving some type of cancer chemotherapy; in a small number the cause for the oversized red cells was not identifiable. It is to be noted in Table 2 that patients with macrocytosis due to liver disease or alcoholism generally were not anemic, or only mildly so. Patients with B_{12} or folate deficiency were more likely to have moderate or severe anemia, although many had little or no anemia. Other

TABLE 3*

DEGREES OF MACROCYTOSIS IN VARIOUS CONDITIONS

	Mean Corpuscular Volume (μm^3)				Total
	115–119	120–124	125–129	130†	
Folate deficiency	14	7	4	6	31
Vitamin B_{12} deficiency	6	3	2	2	13
Both	3	1	1	1	6
Liver disease†	9	5	1	—	15
Alcoholism only	9	2	—	—	11
Other conditions	13	8	3	—	24
All categories	54	26	11	9	100

* From McPhedran et al[4] with permission.

† In the absence of folate or vitamin B_{12} deficiency.

studies[5,6] have emphasized that the MCV increases very early with B_{12} or folate deficiency. In the case of B_{12} deficiency the increase in MCV may occur a few years before the patient has significant anemia, which may result in a serious delay in diagnosis if not recognized and investigated. One study[6] reports that in at least 10% of patients with PA the diagnosis could have been made several months to four years earlier had note been made of an increased MCV with little or no anemia on initial examinations of the blood.

In the group of patients studied by McPhedran and colleagues[4] alcoholism and liver disease were associated with lesser increases in the MCV, rarely over 125 fl (Table 3), whereas larger average red cells were secondary to B_{12} or folate deficiency. Hence, unusually voluminous cells make a diagnosis of B_{12} or folate deficiency likely and other causes less likely. The converse obviously is not true, since the vitamin deficiencies are frequently associated with only slight or moderate increases in MCV, and in fact it is well known that patients with combined deficiencies, e.g., iron and B_{12} or folate, may have a normal MCV.[7]

Undoubtedly it is not so well known that patients with B_{12} or folate deficiency may have a normal MCV in the absence of a concurrent disorder tending to produce microcytosis. In 1979 Prentice and Evans[8] reported a study of 86 consecutive cases of B_{12} or folate deficiency, of whom seven had a normal MCV in the absence of iron deficiency, thalassemia, or any other condition that would tend to normalize mean red blood cell (RBC) size (Table 4). It was their impression that the MCV tends to increase in the megaloblastic anemias, with a decreasing RBC count up to a certain point, but then decreases as the anemia becomes more severe. They were not able, however, to show a statistically significant difference in the MCV between the groups with RBC counts above or below 1.5 million/cu mm. It is apparent from the table that normal MCVs were seen at all levels of the RBC count. In a study of black patients with PA, 42% had a normal MCV.[1] Some patients did indeed have associated iron deficiency and some probably had thalassemia minor, but a significant number had neither of these complications to account for the normal MCV.

TABLE 4*

MCV IN MEGALOBLASTIC ANEMIAS

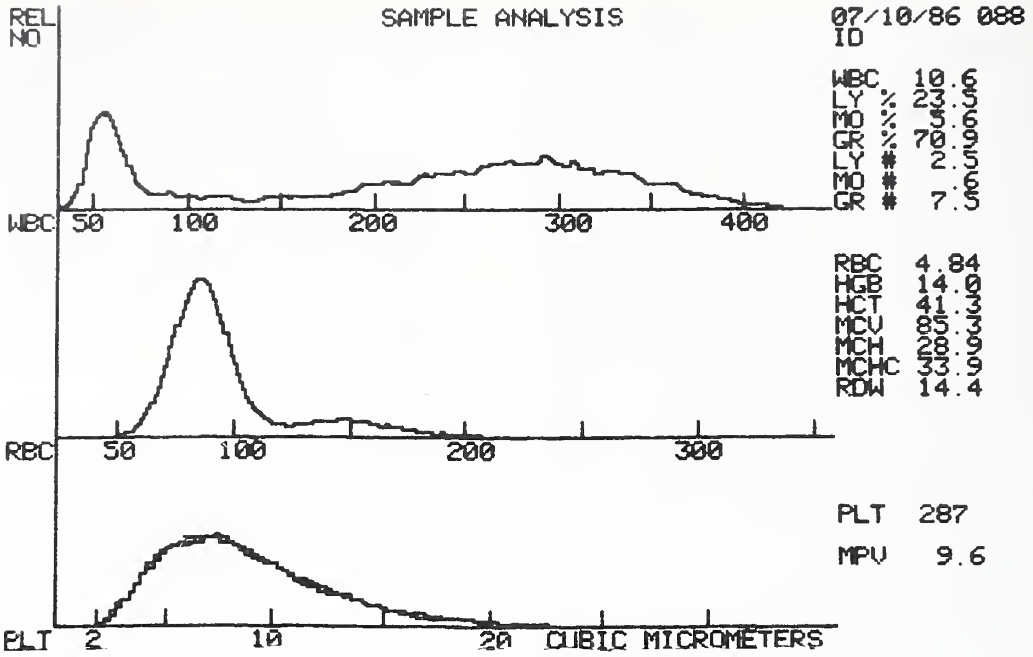
RBC Count (in millions)	No. of Patients	No. with MCV Normal	Mean MCV
>3.00	9	1	109
2.50-2.99	15	2	110
2.00-2.49	16	1	116
1.50-1.99	24	1	119
<1.50	22	2	114

*From Prentice and Evans[8] with permission.

Diagnosis

One might gain the impression from the above observations that the common practice of ordering B_{12} and folate levels routinely in any patient with anemia is justified regardless of the MCV; this certainly is not true and the practice is to be decried. In examining the records of the Immunology Laboratory at the Memphis Regional Medical Center for a one-month period, assumed to be representative, it was found that assays for B_{12} and folate were done on 84 patients at a total cost to them of approximately $4,242. Of the 168 studies done, low values were found in 21, which may or may not have provided useful clinical information. Assuming the 21 low values were helpful in diagnosis, there was then a return of slightly over 12% on the cost of all tests done, which might represent a good return in the world of finance but certainly not in terms of hospital cost containment. It is clear that a means of selecting patients requiring these studies is needed.

The obvious solution, now regrettably neglected, is careful examination of the blood smear. Finding a hyperlobulated monstrosity such as shown in Fig. 1 with a good clinical evaluation of the patient may be all that is required for diagnosis. Various criteria for identifying hypersegmentation of granulocytes have been described,[1] such as one or more cells with six or more lobes, more than 5% with five lobes, or an average lobe number per polymorphonuclear neutrophil (so-called lobe index) greater than 3.5. Hypersegmentation may perhaps be more easily identified by determining the number of bilobed neutrophils since these are easier to recognize than cells with five or more lobes. Normally 20% to 40% of neutrophils are bilobed; less than 20% would therefore indicate hypersegmentation. Using these several criteria for hypersegmentation, it may be expected in up to 98% of patients with B_{12} or folate deficiency.[2] Megaloblastic anemia, however, is not the only cause of hypersegmen-



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WBC   10.6
LY %  23.5
MO %   5.6
GR %  70.9
LY #   2.5
MO #    .6
GR #   7.5

RBC   4.84
HGB   14.0
HCT   41.3
MCV   85.3
MCH   28.9
MCHC  33.9
RDW   14.4

PLT   287

MPV    9.6
```

Figure 2. Normal WBC, RBC, and platelet histograms.

tation, since it may be found in patients with chronic renal disease, and a congenital form has been said to occur in about 1% of whites. Thus hypersegmentation must be considered in conjunction with associated abnormalities in red cells, not only macrocytes, but also macro-ovalocytes, spherocytes, schistocytes, and bizarre often small poikilocytes. As we will point out, the macrocytic picture is not simply that of a single population of large red cells, but a great variety of sizes and shapes.

Additional Laboratory Testing

The later model automated blood cell counters have made available two additional parameters that promise to be useful in the early diagnosis of deficiency states, including both iron deficiency and B_{12} and folate deficiency. The red cell *histogram* is a graphic representation of the red cell size-frequency distribution curve; the *red cell distribution width (RDW)* is a quantitative expression of anisocytosis or variation in red cell volume. Some 60 years ago Price-Jones[9] prepared red cells with a micrometer, plotting percent cells on the ordinate and diameters on the abscissa of a graph. A composite curve of patients with PA was not only shifted to the right (to the macrocytic side) but was greatly widened with a tail of cells extending to the left (microcytic side). He found that patients with PA had a coefficient of variation (standard deviation expressed as a percentage of the mean) in red cell size of 12% compared to a normal of about 6%, reflecting the much greater variability in red cell size in the disease. For obvious reasons the diagnostic utility of his observations has become practical only with the development of automated computerized counters, which rapidly provide the histogram and the RDW. It has been found that the RDW increases early in B_{12} or folate deficiency before there is an increase in the MCV. In conjunction with the blood smear it may alert the physician to early diagnosis. Similarly, the RDW increases (increased variability in RBC size) in iron deficiency before the MCV decreases.

Fig. 2 shows WBC, RBC, and platelet histograms of a patient with normal red cell values—hematocrit 41.3%, MCV 85.3 fl, RDW 14.4% (normal 13.0 ± 1.5%). It is seen that the RBC histogram is a narrow symmetric curve. Fig. 3 shows histograms of the patient presented from a blood study done on the day following admission after transfusion of four units of packed red blood cells. The hematocrit had increased to 29.7% and the MCV reduced to 97.2 fl; the RDW was 27.7%. The peaked portion of the RBC curve represents transfused red cells. Her own cells include not only a prominent macrocytic population extending to the right, but a microcytic "tail" to the left which does not return to baseline at the lower threshold of 36 fl. In fact, a small population of microcytes, less than 20 fl in volume, distorts the platelet histogram which normally returns to baseline well within the upper limit of 20 fl.

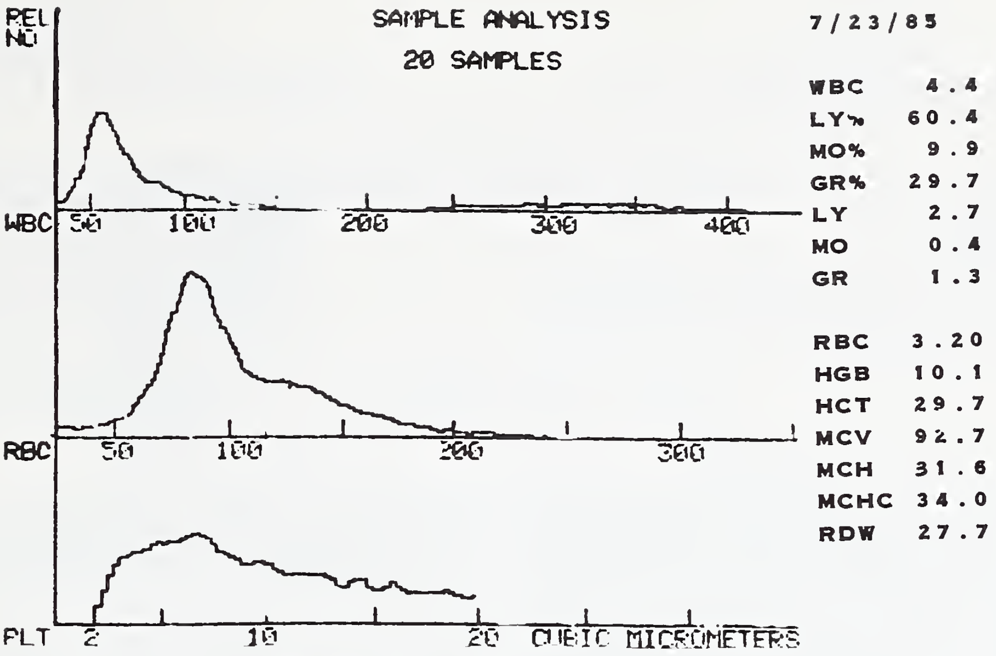

Figure 3. Histograms of patient after four units of packed red blood cells.

Serum B_{12} in Diagnosis

There are several problems with the assay of serum B_{12} which make it less reliable in diagnosis than we would like. One of these relates to the fact that much of the B_{12} in the serum is bound to carrier proteins referred to collectively as "cobalophilins"[10] (Fig. 4). The "nonspecific" binders are transcobalamin I and III, apparently derived primarily from white blood cells or their precursors. B_{12} bound to these compounds is relatively inactive metabolically, and has a half-life of about ten days and perhaps is delivered only to the liver. The "physiologic" carrier protein for B_{12} is transcobalamin II. Only 10% to 30% of the B_{12} is associated with this protein, with a half-life of only ten minutes. Active uptake is from the gastrointestinal tract and storage sites with delivery to tissues where needed or back to storage sites. Thus B_{12} as assayed in the serum includes both the larger but metabolically inactive fraction bound to the nonspecific carriers and the smaller metabolically active fraction bound to the physiologic carrier protein. Patients congenitally deficient in transcobalamin II have a normal serum B_{12} level because of what is bound to I and III but nevertheless have a severe megaloblastic anemia responsive only to large doses of the vitamin. On the other hand, patients congenitally deficient in transcobalamin I have a low serum level, but are not anemic because the concentration and function of transcobalamin II are normal. The myeloproliferative syndromes are characterized by marked increases in the nonspecific binders and in the amount of B_{12} associated with them, masking the development of PA that may occur in these patients.

A technical problem that has been reported relates to the presence in the serum of cobalamin or B_{12} analogs which though metabolically inactive react with binders used in the assay, resulting in spurious normal levels in patients who are deficient. Newer methods using more specific binders supposedly obviate this problem, al-

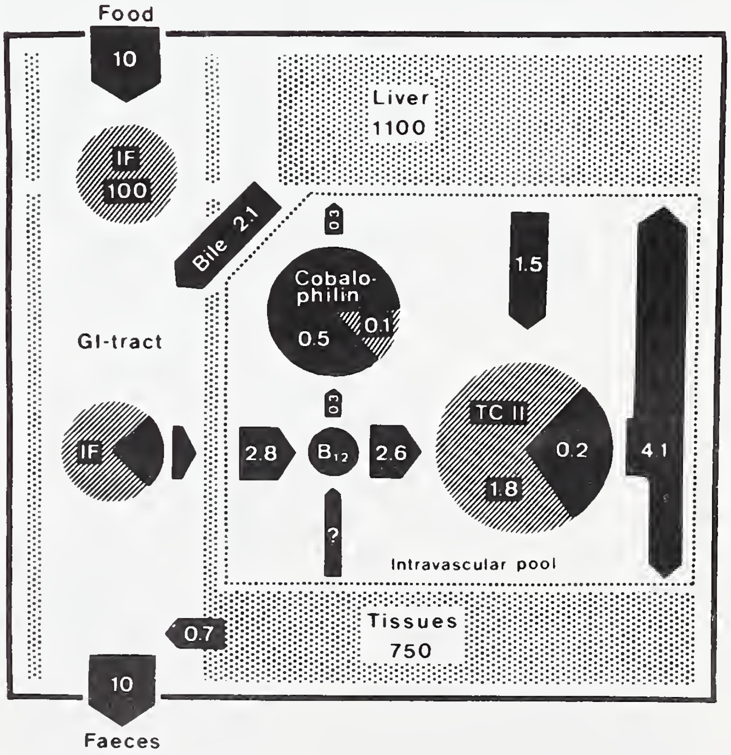

Figure 4. Kinetic mechanism of vitamin B_{12} transport in the body (from Stenman[10] with permission).

TABLE 5*

SERUM B_{12} ASSAY
(Insensitivity—Level Normal, Patient Deficient)

10% to 20% of patients with straightforward deficiency
Nitrous oxide inhalation
Transcobalamin II deficiency
Inborn errors of cobalamin metabolism
Coexistent myeloproliferative syndrome and cobalamin deficiency

*From England and Linnell[11] with permission.

though this is yet to be confirmed.

Problems with the serum B_{12} and folate assays of relevance to the clinician have to do with their insensitivity, i.e., failure to identify all patients who are deficient (false negatives), and their nonspecificity, i.e., low levels in patients who are not deficient (false positives). Table 5 lists some causes of insensitivity of the B_{12} assay, the more serious problem because of missed diagnoses. Ten percent to 20% of patients with deficiency have been reported to have normal levels for reasons that are not clear, perhaps technical.[11] Uncommon causes include inhalation of nitrous oxide, which interferes with metabolism of B_{12} at the cellular level, and inborn errors of cobalamin metabolism. Transcobalamin II deficiency and the myeloproliferative diseases have been discussed above.

Table 6 lists causes of nonspecificity, a less serious problem, at least from the standpoint of patient morbidity; the unfortunate consequence is usually additional expensive laboratory tests.

TABLE 6*

SERUM B_{12} ASSAY
(Nonspecificity—Level Low, Patient Not Deficient)

Folic acid deficiency
Pregnancy, oral contraceptives
Multiple myeloma
Transcobalamin I deficiency
?? Iron deficiency
Interfering agents in serum
    RID† – Isotopes, e.g., gallium-67
    MA† – Antibiotics
        Chlorpromazine
        Antifols†
No apparent cause

*From England and Linnell[11] with permission.
†RID = radioisotope dilution assay; MA = microbiologic assay; Antifols = antifolate antagonists.

Assuming that assays for B_{12} and folate were highly sensitive and highly specific, and if they were ordered selectively only when needed, then maximal information would be provided by three assays: serum B_{12}, serum folate, and red cell folate (Table 7).[2] In true B_{12} deficiency the serum level of the vitamin would, of course, be low. In addition, the level of folate in red cells would also be low because B_{12} is required for entry of folate into cells. Serum folate, on the other hand, would be normal or even high because of methyltetrahydrofolate "trapped" in the serum. This tripartite combination of findings would exclude the possibility of a low serum B_{12} level resulting from folate deficiency.

TABLE 7*

CORRELATION OF B_{12} AND FOLATE LEVELS WITH CLINICAL STATUS

Clinical	Serum B_{12}	Serum Folate	RBC Folate
B_{12} deficient	Low	Normal-High	Low
Folate deficient			
Early—3 weeks	Normal	Low	Normal
Late—17 weeks	Normal	Low	Low
Early repletion	Normal	Normal	Low
Both deficient	Low	Low	Low

*Modified from Herbert.[2]

With insufficient folate intake the serum level drops rapidly, and is low after only about three weeks; tissue depletion occurs much more slowly, such that red cell folate is low only after 17 or more weeks of deficient supply. Hospitalized patients ill for various reasons and taking a poor diet often have low serum folate levels without tissue depletion or megaloblastic anemia. These patients would be identified by doing both serum and cell assays. Contrariwise, the serum folate level rises rapidly, with early repletion either by an adequate diet or by therapy, even though the tissues remain deficient. Such patients may also be recognized by doing both serum and red cell assays. With combined serum and tissue deficiencies, such as may be seen in the malabsorption syndromes, all three assays will show low vitamin levels.

Intrinsic Factor Antibody

Antibodies against gastric parietal cells are found in the great majority of patients with PA, but are also found in a variety of other conditions, so that they are of little value in differential diagnosis. By contrast, intrinsic factor anti-

(Continued on page 35)

Traumatic Laceration of Internal Mammary Vessels

GEORGE O'NEAL VINSANT, M.D. and KIMBALL I. MAULL, M.D.

Most penetrating injuries to the chest are the result of stab wounds or low velocity missiles, and in civilian practice, the majority can be managed successfully by closed tube thoracostomy. Vital signs must be closely monitored and volume deficits replenished, and frequent chest roentgenograms are advisable to determine adequacy of pleural space evacuation.[1] Although intercostal vessels may be injured in stab wounds anywhere in the thorax, precordial wounds may also lacerate the internal mammary artery and/or vein. In such instances, surgical intervention is usually needed.to control persistent hemorrhage and exclude associated injuries to important mediastinal structures.

Case History

After a 16-year-old youth received stab wounds to the left anterior chest below the nipple at the seventh and eighth intercostal spaces he was transported in shock to the University of Tennessee Hospital emergency unit by Lifestar aeromedical service; intravenous fluids and supplemental oxygen were administered enroute. The patient arrived responsive but without recordable blood pressure. When closed tube thoracostomy promptly returned 800 cc of blood, the patient was taken directly to the operating room, where a left anterolateral thoracotomy and exploratory celiotomy disclosed laceration of the internal mammary and intercostal vessels and a lacerated pericardium; the myocardium and abdominal organs were uninjured. Following ligation of the vessels and repair of the pericardium, the postoperative course was without complication, and the patient was discharged on the eighth postoperative day.

Discussion

Eighty percent to 85% of penetrating chest injuries can be managed successfully by conservative methods, including correction of hypovolemia, closed tube thoracostomy, temporary ventilatory support and close observation. Stab wounds are the most common cause of civilian penetrating injuries to the chest. A knife is the usual instrument, though in some areas low velocity handguns are becoming more frequent. In a recent report,[2] the majority of stab wounds involved the left pleural cavity (44%), the remaining injuries being distributed among the right anterior chest (28%), right posterior chest (11%), and left posterior chest (17%).

Some patients with stab wounds to the chest may have minor degrees of hemothorax and/or pneumothorax that may not require tube drainage; Weigelt and Muckart found that delayed pneumothorax and/or hemothorax occurred in approximately 10% of such cases.[3,4] Muckart[4] withholds evacuation of the pleural space in patients with an apical pneumothorax less than 2 cm wide and fluid not extending above the angle of the seventh rib. In the observed patients, only 8% required delayed drainage for hemopneumothorax, the condition most likely to require delayed therapy. Weigelt et al[3] found a similar 9% incidence of delayed pneumothorax or hemothorax in asymptomatic patients with stab wounds to the chest.

Asymptomatic patients without evidence of pneumothorax or hemothorax but with subcutaneous emphysema were admitted for observation. In observing the patient with subcutaneous emphysema, it is important to distinguish the asymptomatic patient without progressive subcutaneous emphysema from the distressed dyspneic patient with expanding subcutaneous emphysema. The latter patient has either a bronchopleural fistula or major lung injury, and may be in danger of developing a tension pneumothorax should the wound in the pleura become sealed.[4]

Persistent hemorrhage is the most common indication for thoracotomy. The hemorrhage is usually measured by chest tube drainage. If chest tube(s) are in place and if bleeding continues briskly enough to cause shock, then thoracotomy

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From the Department of Surgery, University of Tennessee Medical Center at Knoxville.

should immediately be undertaken. Blood return of 1,500 cc following chest tube insertion, continuing blood loss of 150 to 200 cc per hour for three or four consecutive hours, or the need for three or four units of blood to sustain normal vital signs over a 10- to 12-hour period are other indications for surgical intervention. Penetrating injuries to the heart, pulmonary hilum, or subclavian artery will also require early surgical intervention.

In stab wounds to the chest, the most common vessels to be injured are the intercostal artery and/or vein, which together with the internal mammary artery account for over half of the traumatic hemothoraces. Penetrating wounds to structures at the thoracic outlet are very hazardous. The patient may initially appear stable, only to exsanguinate from an unsuspected vascular injury. Pulsatile bleeding, or hematoma with differential pulse in the arm or underlying systolic bruit all indicate major vascular injury, and require an arch aortogram, though only if the patient is stable.

The placement of a chest tube in the patient with major vascular injury must be done with care, as the release of the tamponade from the hemothorax may exacerbate hypovolemic shock. Repair of the injured vessel should be done early; delayed hemorrhage, arteriovenous fistula, and pseudoaneurysm complicate the procedure.[5] Penetrating wounds to the heart require immediate operative intervention, since primary treatment with pericardiocentesis has an associated mortality of 35% for stab wounds and 40% to 100% for gunshot wounds. Pericardiocentesis should be used only as a temporizing measure while the operating room is being set up for the patient who reaches the hospital alive.

In addition to persistent hemorrhage, thoracotomy may be required for massive air leak or esophageal perforation. The morbidity and mortality associated with esophageal perforation is high, particularly if diagnosis is delayed; morbidity is higher with thoracic esophageal perforation, and the mortality may reach 67%.[6] Mediastinal penetration should alert the clinician to the possibility of esophageal injury, especially with firearm wounds. Likewise, penetration of the intrathoracic trachea or a major bronchus should be suspected when there is massive intrapleural air leak and progressive tension pneumothorax. Penetrating wounds of the lower chest may be accompanied by intra-abdominal injuries in 10% to 30% of patients; mortality is significant and increases with the number of organs injured.

Summary

Patients with penetrating trauma to the chest can usually be managed by closed tube thoracostomy, fluid replenishment, and close observation. Early thoracotomy will be required in 10% to 15% of patients with persistent hemorrhage.

Precordial wounds may injure the internal mammary vessels and cause exsanguinating hemorrhage. In addition, mediastinal structures are in jeopardy, including the heart, trachea, bronchi, and esophagus. The surgeon must be ever aware of these potentially lethal injuries, and be prepared for immediate operative management.

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Radiology Case of the Month

Asymptomatic Anterior Mediastinal Mass

JOSEPH G. JACKSON, M.D. and STEPHEN L. GAMMILL, M.D.

Case Report

As part of an otherwise normal preemployment physical examination, an 18-year-old white man obtained a chest radiograph at our facility (Fig. 1). The abnormal right heart border prompted further evaluation, including a lateral chest film (not shown), which demonstrated obliteration of the retrosternal clear area. CT scanning of the thorax (Fig. 2) revealed an extensive anterior mediastinal mass with mixed attenuation characteristics. On more cephalad images the mass did not appear to extend into the superior mediastinum. Except for a minimally elevated alkaline phosphatase, laboratory studies, including a CBC, thyroid, and liver profiles, were normal. The patient denied any symptoms except for vague "chest tightness" some two weeks previously. Mediastinoscopy was performed and tissue for diagnosis was obtained.

Please examine Figs. 1 and 2 and choose the best diagnosis:

(1) Thymoma
(2) Teratoma
(3) Substernal thyroid
(4) Hodgkin's lymphoma

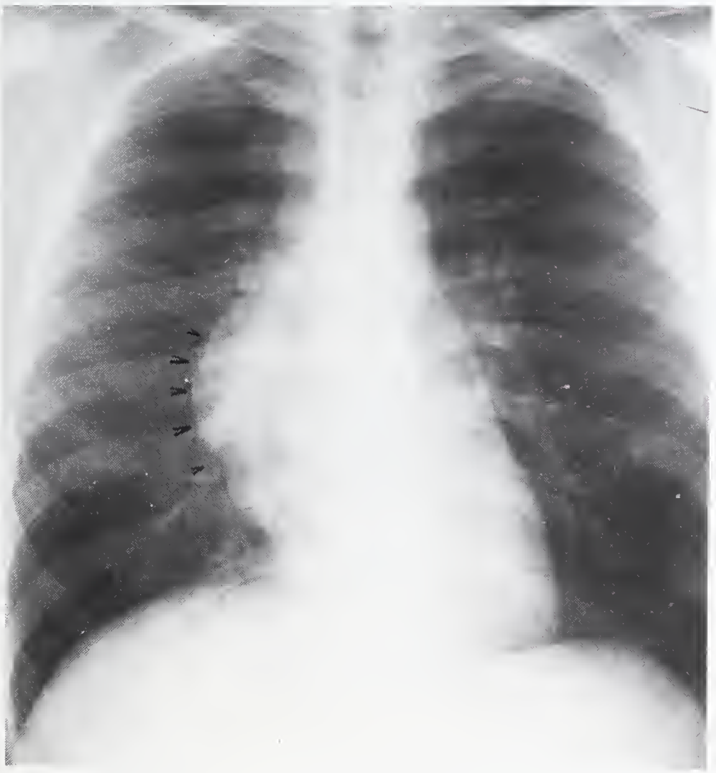

Figure 1. PA chest radiograph demonstrating abnormal right heart border (arrowheads).

Discussion

Of the numerous entities that can result in a mediastinal mass, the listed possibilities account for the majority of *anterior* mediastinal mass lesions. Each has radiographic and clinical characteristics that often make a more precise diagnosis possible.

Thymomas can be detected at any age, with a peak incidence during the fourth decade of life. They may be either benign or malignant, and there is a close association to myasthenia gravis. Up to 50% of patients with thymomas have stigmata of myasthenia, while only about 15% of patients with myasthenia are found to have thymomas. The lesions may be cystic or solid, and calcifications are noted in some cases.[1-3]

Mediastinal teratomas occur about as frequently as thymomas, most of them in teenagers

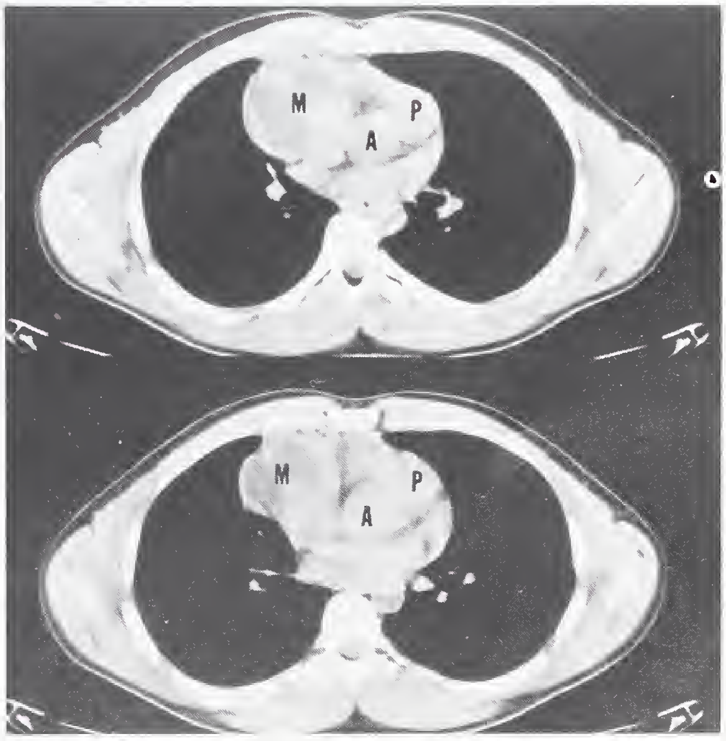

Figure 2. CT scan of the midthorax demonstrating a mixed attenuation, anterior mediastinal mass (M = mass; A = aorta; P = pulmonary outflow tract).

From the Department of Radiology, Baptist Memorial Hospital, 899 Madison Ave., Memphis, TN 38146.

and young adults. Cystic and solid forms have been described, the former most commonly benign and the latter often malignant. Malignancy occurs in approximately 25% to 30% of cases. As with pelvic teratomas, various combinations of ectodermal, endodermal, and mesodermal tissue make up the lesion. The CT appearance depends on the composition of these various elements. High concentrations of fat or sebacious material would result in negative CT numbers. Approximately 50% of the tumors have calcifications, but it is uncommon to detect formed osseous or dental elements within the mediastinal forms; they are pathognomonic if present.[3,4]

Mediastinal thyroid tissue accounts for about 10% of resected mediastinal masses and almost always originates as an extension from the cervical thyroid. These endothoracic goiters can occur anywhere in the mediastinum, but most often are found in the superior, anterior region. Tracheal deviation and compression is an important finding on chest radiographs and there are several important CT findings that have been reported to help in diagnosis. Probably due to its iodine content, thyroid tissue demonstrates CT numbers higher than surrounding soft tissues. Due to its affinity for iodine, the tissue will take up iodine-rich contrast material and prolonged enhancement will result. Detection of continuity with the cervical thyroid is a key to diagnosis, but it may be missed without careful attenuation to technique (i.e., extending the scan into the neck). Calcifications are frequently demonstrated. As an adjunct, radionuclide scanning with iodine-131 will localize thyroid tissue in nearly all cases.[5,6]

Hodgkin's lymphoma, the largest group of malignant lymphomas, has a bimodal peak incidence, occurring in the third and in the sixth through eighth decades of life. A lobulated mediastinum is often the presentation on chest radiography. Calcifications are rare but can be seen in tumors of long standing, or following radiation therapy. The CT numbers are usually lower than soft tissue and the lesions often contain cystic or necrotic areas. The vascular supply is poor and contrast enhancement is limited.[5]

In the case at hand, an asymptomatic anterior mediastinal mass was detected on chest radiographs. The patient had no clinical findings suggesting myasthenia gravis. CT scanning confirmed the findings on chest radiography, but failed to demonstrate calcifications and there was no contrast enhancement. The lesion did not appear to extend into the superior mediastinum or neck. Attenuation factors were mixed, suggesting cystic change or necrosis. A presumptive diagnosis of lymphoma was subsequently confirmed by mediastinoscopy.

FINAL DIAGNOSIS: Hodgkin's lymphoma.

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HELP FOR IMPAIRED PHYSICIANS

Through its Committee on Impaired Physicians, TMA helps doctors who are suffering from alcoholism and drug addiction. The thrust of the program is rehabilitative, not punitive. The Committee is composed of physicians who have special expertise in these areas, some from personal experience. Effective treatment for these illnesses is achieved most easily when the disease is detected early and family, friends, and associates are urged to avoid misguided sympathy which enables the condition to deteriorate.

HELP US TO HELP

Call the TMA Impaired Physician Program (615) 327-2711; outside Nashville call collect. Phone service available around the clock.

MRI Case of the Month

MICHAEL C. GAROVICH, M.D.; CHARLES L. ROBINETTE, JR., M.D.; and
FRED CALLAHAN, M.D.

Case Report

A middle-aged white man had multiple vague complaints including headaches, parathesias, and intermittent history of difficulty in walking and lack of upper extremity coordination. Can you make the diagnosis from Figs. 1 and 2?

The MRI scan demonstrates multiple lesions in the periventricular white matter indicating high signal intensity. There is no evidence of mass effect. These findings are strongly suggestive of multiple sclerosis.

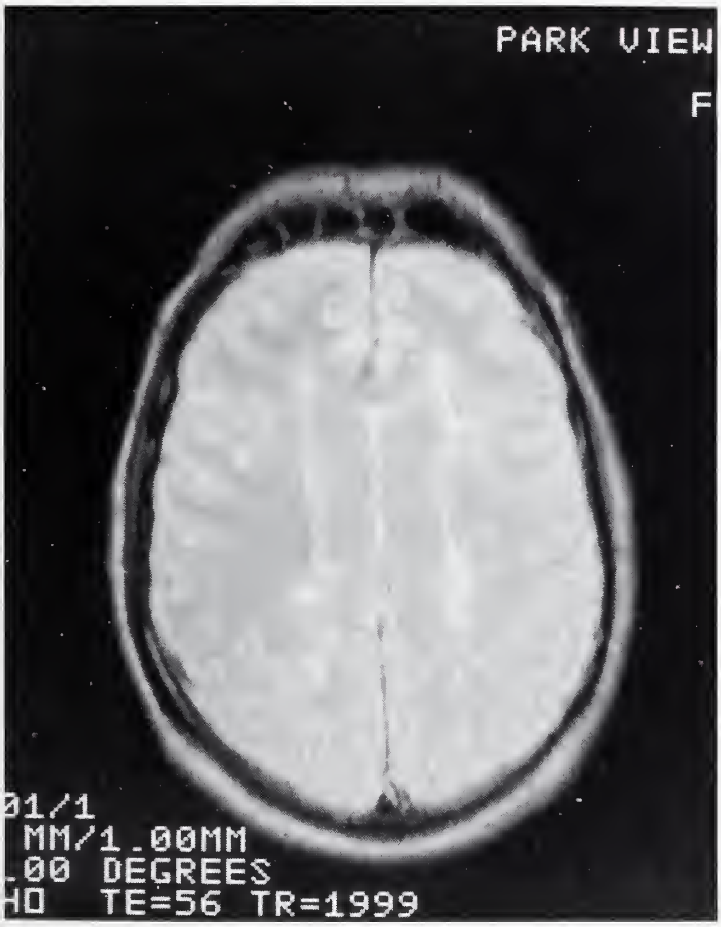

Figure 1. This intermediate T_2 image demonstrates multiple high intensity (white) lesions in the periventricular white matter. In this sequence the ventricles are isointense with the normal white matter.

Discussion

Multiple sclerosis produces various clinical signs and symptoms and is recognized by recurrent relapses and remissions. Individual lesions are related to plaques of demyelination. MRI, due to its inherent properties, is extremely sensitive in differentiating gray from white matter, and white matter abnormalities. It is therefore the imaging procedure of choice in demonstrating multiple sclerosis plaques and other degenerative or inflammatory states affecting white matter. CT is frequently normal in these situations.

DIAGNOSIS: Multiple sclerosis.

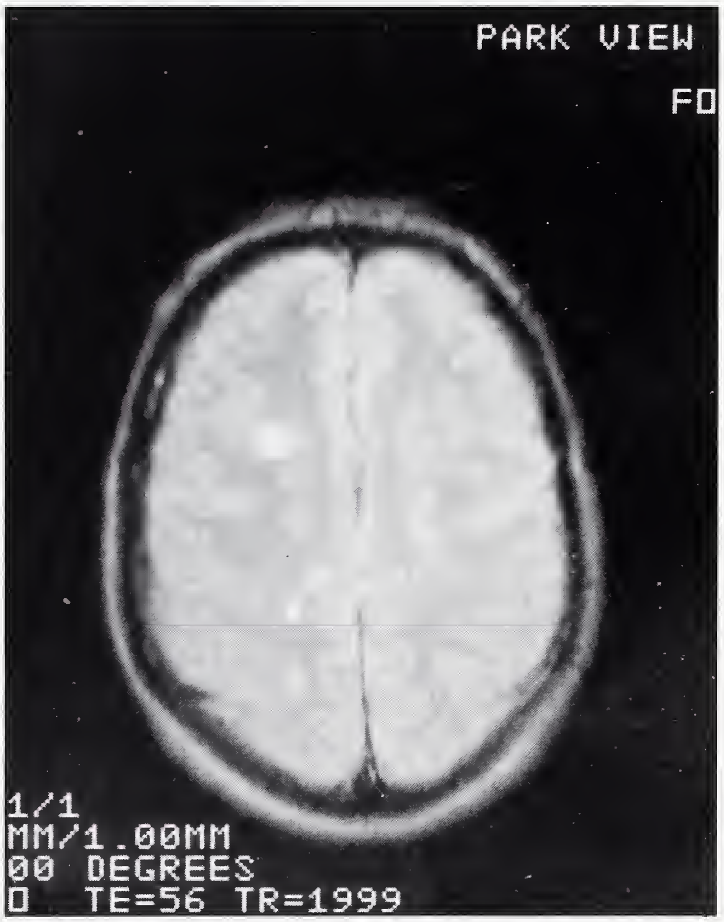

Figure 2. Additional plaques (high intensity) appear in the deep white matter of the corona radiata.

From the Departments of Radiology (Drs. Garovich and Robinette) and Neurology (Dr. Callahan), HCA Park View Medical Center, Nashville, TN 37203.

The Tennessee Communicable Disease Bulletin

ROBERT H. HUTCHESON, M.D.

Introduction

The Communicable Disease Bulletin published by the Tennessee Department of Health and Environment may be familiar to many TMA *Journal* readers because it has been mailed monthly to physicians and hospitals since October 1975. This four-page bulletin is one way the Communicable Disease Section of the Tennessee Department of Health and Environment communicates with physicians. Each physician with a Tennessee address on the licensing board's mailing list receives the "little yellow sheet" each month. This brief article will outline its history, origins, purpose, evaluation, subject matter, and potential for the future.

History/Origins/Purpose

In the early 1970s before becoming a monthly letter, the first issues of the Communicable Disease Bulletin began appearing in physician offices in Tennessee. Other states were beginning or had just begun similar ventures for the same general reasons: the states with communicable disease sections typically request physicians to assist with disease reports and other data so that these communicable disease sections can carry out their work. But what did physicians and hospitals receive in return? A typical answer was that large numbers of physicians saw none of the results of their reports or other data. Most communicable disease workers believe that a request for data, especially when the request goes beyond the narrow confines of law, creates an obligation to let readers know what was done with their reports. Hence the several states began to publish their version of the Communicable Disease Bulletin. Tennessee's first efforts were sent on an irregular basis during the first half of the 1970s. During 1974 for example there were three issues, not 12 issues as there are now. We reasoned that we could wait "until we had something to report" and would thus save postage. At that time we did not know first class rates were about to jump, and that we, unlike most organizations, could obtain postal savings intended for newspapers and journals by becoming one or the other. We did not know that our small section would always have something to report. That "something" was what was happening, what we were planning, and new developments in infectious disease that might be missed. But which of the three ought we emphasize?

Evaluation

We get mail and attend medical meetings. Physicians call us. Based on these contacts we learn what the physician reader will tell us about the Bulletin. We listen and try to repeat what "works" based upon these contacts. We have tried tables, graphs, charts, and graphics without a single comment in over ten years. It is the narrative, not the visuals, that receive favorable comments. So we concentrate on narrative, using graphics sparingly. As for content, physicians continually tell us that disease events in Tennessee are what they like. Diseases, then, are our emphasis.

Subject Matter Highlights from Previous Years

Measles in 10 to 16 year olds in Friendship, TN [Dec 74]
Nosocomial illness from Zepharin solution [June 75]
St. Louis encephalitis returns [Oct 75]
Leptospirosis in Stewart County [Nov 75]
Tetanus [Feb 76]
Swine flu program [July 76]
First case report of infant botulism [Jan 77]
Your editor gets shigellosis [Feb 77]
Rubella epidemic [Apr 77]
Brentwood epidemic pneumonia—Histoplasmosis [July 77]
New school immunization law [June 78]
DTP Lot 64-201 withdrawn [Apr 79]

From the Tennessee Department of Health and Environment, Nashville.

Review of deaths from venomous animals in Tennessee [Oct 79]
Paraquat poisoning presents as diphtheria [May 80]
365 ill persons in two foodborne outbreaks [June 80]
Marinated cucumber in mineral spirits and glycol ether [July 80]
Pseudomonas folliculitis in a swimming pool [Jan 81]
Rubella at UT Martin imported from Japan [Oct 81]
Epidemic kerotoconjunctivitis [Nov 81]
1981 highest rabies year in the past ten [Feb 82]
Staphylococcal cellulitis among river guides [Nov 82]
Airborne nosocomial varicella [Apr 83]
AIDS in Tennessee [July 83]
Mowers tularemia (poor bunny, poor mower!) [Aug 83]
Ocular Candida parapsilosis in Tennessee and elsewhere [Jan 84]
AIDS in Tennessee [Sep 84]
Child restraint device effectiveness in Tennessee [Nov 84]
Influenza in Tennessee [Feb 85]
Rocky Mountain spotted fever in Tennessee [Apr 85]
Alternative testing sites for HTLV-III in Tennessee [June 85]
Animal rabies in Davidson County [Oct 85]
Syphilis in prisons [Jan 86]
Chlamydia in Tennessee pregnant women [Feb 86]
Meningococcal disease in Southeast Tennessee [Mar 86]
Febrile illness in a college fraternity [May 86]

Future Plans and Summary

Like other states, Tennessee will probably continue reporting monthly those disease events of most interest to physicians. The previous 12 years have been ones of changing diseases. Infectious diseases will vary in their specific influence. When new diseases appear, when old ones reappear, we hope to report them to you through the Tennessee Communicable Disease Bulletin.

Medical Grand Rounds . . .

(Continued from page 28)

body, which is present in almost 60% of patients with PA, is rarely found in other conditions, examples being relatives of PA patients or patients with thyroid disease. Therefore, the finding of intrinsic factor antibody in a patient with megaloblastic anemia and a low serum B_{12} level may be considered diagnostic of PA; this recognition may represent a considerable saving in time and money for the patient, since the cost of the antibody determination is about $60, whereas confirmation of the diagnosis by parts I and II of the Schilling test costs approximately $370.

Acknowledgment

We thank Miss Ann Bell, M.S., for her help in preparation of the manuscript.

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Loss Prevention Case of the Month

Preventive Loss Prevention

J. KELLEY AVERY, M.D.

Case Report

Dr. O, an orthopedic surgeon, and a 62-year-old colleague, Dr. N, a neurosurgeon, both on the staff of Middletown General Hospital, attended the same medical meeting in a nearby city and the two of them had dinner together the first evening of the seminar. Dr. O noticed that his friend, Dr. N, had the odor of alcohol on his breath when they met in the hotel restaurant. Dr. N had three drinks while waiting for dinner, selected the wine, and insisted on brandy following dinner. It was a very enjoyable evening and there was no indication that Dr. N was intoxicated. They parted agreeing to meet the following morning for the first session in which they each had expressed a special interest.

The next morning Dr. N did not appear for the session as agreed, and Dr. O called his room thinking he had overslept. Dr. O heard a clatter on the other end of the line as if the phone had slipped when picked up. Dr. N answered with slurred speech and seemed incoherent. Dr. O told him not to try to get up, summoned the manager, and went to his friend's room where Dr. N was found dressed and lying in bed. Dr. N related an episode of unsteadiness during the early morning hours while going to the bathroom, but he returned to the bed with no problems. Upon arising, he bathed, shaved, and dressed, but felt nauseated, vomited, and returned to bed.

Dr. O's cursory examination revealed a normal blood pressure, pulse, and respirations. Gross neurological signs were within normal limits, but Dr. N continued to be ataxic and displayed a definite memory disturbance, asking "where am I?" and "how did I get here?" Dr. O took him to the emergency room of the local hospital where a neurologist friend of Dr. O's examined him thoroughly and advised hospital admission for further observation and study. Dr. N begged off, having become totally asymptomatic; he promised to return home immediately and undergo the studies as suggested. Dr. N's wife drove to get him and they returned to Middletown.

After the meeting Dr. O returned home and on the following Monday learned that Dr. N had not entered the hospital or consulted the specialist as he had promised to do. When Dr. O saw him, Dr. N stated that he had diagnosed his episode as being related to the seafood he had eaten at the restaurant and recalled a similar episode in the past.

About two weeks later Dr. O was discussing the medical meeting with Dr. N's partner, Dr. M, and mentioned the episode. Dr. M remarked that Dr. N had another episode the past weekend and had not covered for Dr. M on Friday evening as promised and made rounds on Saturday night after 10:30. Dr. M mentioned Dr. N's recent move from home into an apartment.

What should Dr. O do?

- Confront Dr. N with his suspicions.
- Look at his surgical performance for the past year, his postoperative complications, etc.
- Make a confidential report to the chief of neurosurgical service.
- Report concerns to the TMA Impaired Physician Program.
- Do nothing.

Loss Prevention Comments

The case you have just read has happened over 300 times in the past eight years in Tennessee, as documented by the Tennessee Medical Association's Impaired Physician Program. To all the Dr. O's in Tennessee, the 10% who are Dr. N's need your attention, concern, and love. You can express all these in an effective way by enlisting the assistance of the Impaired Physician Program. Over 95% of the cases, where the TMA program has intervened, have returned the physician to his practice, preserved his liability coverage, and maintained his individual health. Call the TMA Impaired Physician Hotline at (615) 327-2711; outside Nashville call collect.

Dr. Avery is medical director for Continuing Medical Education, St. Thomas Hospital, Nashville.

James R. Royal

Resolution #1—1987
Indigent Care

As we begin the new year—1987—we must address the ever-increasing problem of indigent health care. Governor McWherter has placed this high on his list of priorities for this year. I believe we can and must be a part of the solution. Since the government alone cannot provide the care, it is up to us to deliver it in the proper quantity and quality. They can allocate the resources to ensure its availability. The present Medicare and Medicaid systems have not done the job. They've only helped contribute to massive deficits and debt.

Most of us, and generations of physicians before, and most hospitals have always provided needed medical care regardless of the patients' ability to pay. Reduced and no charges have always been around and unpaid bills are a fact of life. It has been estimated that at least 6% of fees are never collected and another 9% of potential fees are reduced. Is this enough contribution by the medical profession to the indigent health care problem?

A certain segment of the population has "fallen through the cracks" in regard to having adequate protection against health care costs. These are people whose age or economic status preclude their eligibility for protection under tax supported programs, but who find it very difficult to purchase coverage in the private sector. Some are uninsured all or part of the year and some are underinsured. These are usually women, part-time employees, self-employed, unemployed, uninsurable persons in poor health, and other low income workers. Many of these and their dependents have no access to coverage and must seek health care wherever they can find it—usually in clinics and emergency rooms of public hospitals—the most expensive way to provide care.

We have provided basic public education from day care centers through postgraduate programs, but not everyone participates through all levels. For various reasons many drop out at different stages. The public housing system is another example of a multi-tiered tax supported program. It has been decided that the governor should reside in a "mansion," that certain college presidents and hospital administrators should live in luxurious homes, whereas others, the elderly and the poor, be provided with public housing projects. The pecking order is very evident in housing and services on any military base and in all levels of government. Maybe a single-tiered health care system is not the answer.

No other group has benefitted more from the public education system than the doctors of Tennessee. Thousands of Tennessee physicians have attended colleges and medical schools at taxpayers' expense. Many of us could not have afforded the private schools. Surely, we can help the governor and legislature devise some method of providing basic health care for all our people without falling into the "socialized" trap—even if we have to give up some of our income. With the ever-increasing "glut" of doctors, I don't believe we will be as busy as we have been in the past and can devote a little more time to indigent care.

The government cannot provide health care. They can only promise it and decide how much to pay for it. We must deliver it. I propose that we join with our government and do it.

James R Royal, MD

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JANUARY, 1987

editorials

Survival in One Not So Easy Lesson

The true Renaissance man was characterized by his vast knowledge of many—indeed every—subject; more than that, he was characterized by his curiosity, and an avidity for satisfying it. It was this spirit of inquiry that was the hallmark of the Renaissance; a lot of icons were smashed in the process, among them inevitably some that did not need smashing. In earlier days a bright man could know, if not everything there was to know, at least a good part of it. That spirit of inquiry set off an information explosion that has not abated after some six centuries, and in fact continues compounding itself geometrically.

Knowledge builds on knowledge, and the more answers those inquisitive minds found, the more questions they found to explore. All sorts of frontiers continued falling. By the time I reached medical school, our teachers were beginning to tell us, with what seemed to us blatant hypocrisy when exam time rolled around, that as it was impossible to know everything, the appropriate response to such a vast information base was to know not necessarily all the answers, but where to locate them quickly in time of need. They were assuming a certain basic core of knowledge; what that basic core consisted of set faculty at odds with one another in the days following World War II, and saddled curriculum committees with an impossible and thankless task. It led to fragmentation of the curriculum, leading I suppose to the increased use of the term curricula, since there clearly was no longer only one.

It got even worse. It changed the way medicine was practiced, too. A generation of specialists was bred by the military service in response to the demands of war, and young men returned in droves to fill residency spots under the GI Bill of Rights; whereas once the well-trained specialists had been pretty much confined, if not to academia, at least to metropolitan hospital centers, they now appeared in the hinterlands, working in newly constructed, federally funded community hospitals.

Some of the general practitioners who had been the sole proprietors of medicine in the smaller communities adapted by changing their practice patterns to accommodate to the influx of specialists. Others survived as anachronisms, with the result that their patients received barely, or even sometimes less than, adequate care. Mercifully, those were few. Though scientific medicine flourished, its art languished, and with it the doctor-patient relationship, bearing within it some of the seeds of a litigious spirit waiting in the wings, and now upon us.

Through all of this, medical students had come and gone, giving scant attention to either the medical or lay establishment. That was until

Vietnam. That so-called non-war, and other distressing events that preceded it, sired a rebellious youth in this country; rooted in the maladjustment of those days is our present drug culture. The students of that period paid a lot of attention to the establishment; wherever one existed, they sought to dismantle it and rebuild it in their own image. They assaulted the halls of organized medicine and the medical schools, demanding a voice in determining their own future, including deciding what courses they should take.

That their demands were not without merit was annulled by their approach; it threatened to abolish the hard won gains already accomplished by students and residents to form a section in the AMA, for example, as the great unwashed and bewhiskered descended upon and attempted to disrupt the sessions of the House of Delegates.

Today's radicals are tomorrow's conservatives, and as some of those activists became members themselves of the establishment, they incorporated into it through the subtler but more effective means of both persuasion and the vote many of the better things they had attempted by revolution. There are now medical student members at all levels of the Federation, and both the resident and student sections have members on the AMA's councils and Board of Trustees. They are serious about their responsibilities, and have made valuable contributions in deliberations; they have gained both the attention and the respect of their older colleagues.

Things change, and survival depends on the ability to adapt, frequently to an inhospitable environment. Medically speaking, after the initial antiscientific flap of the flower children generation wore off, the profession has had infused into it a better, or perhaps more precisely, a renewed understanding of the physician-patient relationship, of the sort that the physician of pre-World War II days had, but which got lost, or at least misplaced, in succeeding decades.

The winds of change have kept on blowing, and indeed seem presently to be accelerating. Those of us who once had trouble with the "slipstick" are now being confounded by computers. We are finding out that not only is it not possible these days to know everything, or even to read—or peruse, even—everything in one's own field, let alone medicine in general, it is also not possible to look the answers up without being able to ask the right questions—of a computer, yet! So now that is what students are being taught.

Adaption to such necessity is not what is difficult for the physician; medical progress and patient welfare require it. It is the changes in medical practice patterns and the external pressures from society that cause dislocations in the physician. It is hard on a physician to have to accommodate to being treated as a tradesman; it is hard to accommodate to the PPOs and HMOs, and to the progressive litigiousness of society, fueled by the contingency provisions of the tort system. Some of our colleagues have given up the fight, and the number of good college students who are entering medicine is dwindling. It is harder still to be told by a third party how we should care for our patients. The pressures take up time and energy that the physician could better use for his patient. The regulations are stultifying, strangling initiative, and ultimately also working to the harm of his patients.

The reasons for entering medicine as a career are various, but enter it individuals always have, even in the days when to lose a patient meant to lose one's head. The human being generically is a remarkably adaptable creature. Individually though, whereas some, maybe most, are resilient, others are brittle, and some are simply pliable. A tree that can bend with the wind will not break, and sooner or later the wind will change and it will bob up again.

The motto of the Seabees, the U.S. Navy construction battalions of World War II, was, "The difficult we do immediately; the impossible takes a little longer." We enter 1987 faced with obstacles to the continued care of our patients that often seem insurmountable; just because no solution is readily available, though, does not mean that none exists.

Predicting the future is chancy at best. I would simply point out by way of a reminder that a couple of decades ago Medicare was going to destroy medicine. Now we hear it is the abandonment of it that will. Perhaps the original assessment was correct after all, since if Medicare had not begun, we would not be faced with its ending, and we also would not be faced with such things as fee freezes and assignments. Then again, perhaps we would; if it had not been Medicare, it would almost certainly have been something else.

It all goes to prove that there are few safe harbors, and no certain ones; what's more, there never have been. Don't expect to find any this year, either.

Happy New Year. Illigitmi non carborundum.

J.B.T.

Kick 'em While They're Down

All the while that we are bemoaning our situation these days between the rock and the hard place, we can, and do, do a lot for the sick. If I had to pick out a single advance since my medical school days that surpasses all others in saving lives, it would be the discovery and development of antimicrobials. It really began in my high school days with the discovery of sulfanilamide, but the real revolution began with the advent of antibiotics.

We have generally become so blasé about infectious diseases that I am moved from time to time to point out that pathogenic microorganisms, while they may be down, are far from out. They still can, and frequently do, kill, even under the best of circumstances. I thought about this as I wrote about adaptability, of which germs are a remarkable example.

Almost any medical publication that you pick up, and often lay ones, too, is witness to their perseverance. The *Morbidity and Mortality Weekly Report (MMWR),* published by the CDC, documents their ubiquitous presence among us. For instance, last week *MMWR* reported 12 cases of cholera in Louisiana, thought to have been transmitted through contaminated shellfish. Since shellfish are widely transported these days, such organisms may be disseminated to inland areas and make their presence known with an explosive watery diarrhea, without ever being detected. With proper hydration, the disease is self-limited, but its course can be mitigated by tetracycline. Without proper treatment, the victim can die. Pearl: boiling a crab for 8 minutes will not kill the vibrio; 10 minutes will.

The gonococcus has remained remarkably durable as a pathogen, despite our early elation at the prowess of penicillin in the war against V.D. (now referred to as STD—sexually transmitted disease—possibly in deference to the concept of separation of church and state). Its incidence increased last year for the first time in ten years, due partly to resistance to therapy and partly to cutbacks in government funding of clinics, as the major portion of funds for STD research and development is being diverted to the AIDS epidemic.

From the Kentucky Department of Public Health Services comes word of two deaths in children from *Baylisascaris procyonis,* a roundworm widely enzootic in raccoons. The worm's larvae produce a visceral larva migrans, leading to an eosinophilic meningitis for which there is no treatment. Since almost all raccoons are infested, and can shed 100,000 eggs per gram of feces, one needs to steer clear of the cute little rascals. A young one can make a delightful pet; it is also a loose cannon in the kiddie-coop. The worms do not know where the Kentucky-Tennessee line is. So oust the beasts.

I could name you a bunch more, but you get the idea, and in fact you will have seen some in your own practice, probably on most days. The interesting thing is that except for the "common cold" (if there is any such thing) diseases due to microorganisms are generally preventable, given sufficient interest and education on the part of the intended victim, and assiduous attention by the health department.

That last statement is pregnant with both possibilities and snares. A properly funded public health system can work wonders. We do not at any level have a properly funded public health system; it is a prime target for budget snipping, and snipped it has been. The *MMWR,* for instance, was once sent free for the asking to any physician. That gold mine of information will now suck up your gold to the tune of a couple of C-notes a year. Fortunately for us, government publications cannot be copyrighted, and so the reprinted publication can be subscribed to from the Massachusetts Medical Society for a paltry $25. It is also summarized in *JAMA*.

As to the intended victims, it takes more than a heap of education to impede gratification of the hunger and sex drives, as any country boy or girl knows, and the city ones at least suspect, I expect. Preventive measures have a low priority under certain circumstances, but even a low priority is better than no priority, which is what the uneducated necessarily have.

Failing education, the next step is case finding and reservoir eradication. That can be done, because it has been with rabies and smallpox, and to a large extent with tuberculosis. That was done in the days when preventive medicine was receiving more than lip service, though. Case finding through cluster investigation needs to be done for a lot of things other than AIDS.

In the 1930s Al Capone was public enemy number one, but there were also a lot of other dangerous gangsters around and the FBI had to broaden its scope. AIDS is now considered public health enemy number one, and it should be. But just as Al Capone had lots of dangerous company, so has AIDS. While we are going after the big game with an elephant gun, we need to keep at least a shotgun handy for the smaller game; it is of no use, either, if it isn't loaded.

J.B.T.

Song of the Islands: Reprise

It's just that things are different in the Islands. For instance, the AMA House of Delegates customarily begins its sessions at nine o'clock in the morning, the opening session excepted, which begins at two o'clock on Sunday afternoon. When the House meets in Honolulu though, which it does every five or so years, things start up at 7 AM, and the first session opens at 11 AM Sunday; this change is made so that everybody can get out and play in the sand and sun. It's all very well to spend your days in a windowless hall when the meeting is in Chicago, but when you have traveled five or ten thousand miles to be in Paradise, you handle things differently. (Not that Honolulu is exactly Paradise, but it's close by, and Waikiki isn't bad.)

When at the opening of the House in December 1979 it came time for the invocation, it was church time. The minister commented that he was retired from active duty, so that he was available; only such as he, he said, could, after all, invoke Divine blessing if the House insisted on opening at such an hour on Sunday. The reason I mention this is a long story—not one that takes a long time to tell, so don't go 'way; I mean it begins a long time back.

A few hours after I graduated from medical school on Sept. 11, 1944, I stood with my bride before the Reverend William Phifer, minister of the Westminster Presbyterian Church in Nashville, and heard him pronounce us man and wife. The next thing I heard Bill Phifer say was the remark I have just recounted. Right after our honeymoon, we left Nashville for my internship in Columbus, Ohio, and a short time before we returned five years later he left for Kansas City. Some years after taking another pastorate in Los Angeles, he was called to the First Presbyterian Church in Honolulu, where he retired in 1974 and has made his home since. On that visit to Honolulu my wife and I spent a pleasant couple of hours with the Phifers in their home, renewing an acquaintance that had lapsed for nearly 40 years. That was my island year; my wife and I had been to Tahiti in May and I to Honolulu (and Oahu generally) and Kauai in October. We went to Maui and the Big Island on a post-meeting tour on this trip.

Though they can't do it much anymore, and actually it has been considered bad form within my memory, preachers used to be big on accusations of whoring after false gods, a thoroughly Biblical expression. This always used to stir up in me visions of the child-eating Moloch, and Dagon, the fish god, and maybe Jezebel—you know—gross things; those aren't long on seduction. The dangerous false gods are more goddesses, actually—the likes of Delilah and Salome, whose blandishments held out an offer of a real bacchanal—like Venusburg for Tannhauser, say. (Whoring after anything much wasn't then open to the ladies, which accounts for the sex bias here as in Biblical times. They do have the same options and proclivities now, though.)

I hadn't thought much, if at all, about Bill Phifer during the intervening years until my wife said he was returning for a homecoming at Westminster last Sunday (Sept. 18). So we met again, and had a brief visit, with recollection of those two previous occasions. Now, though I may not have given Bill Phifer much thought, I have thought about his Islands a lot. I'm not sure I would like living there, but I wouldn't mind giving it a try. The major disadvantage—in fact, the only one I see—would be in having to forsake Middle Tennessee. I often look at pictures I have taken on my visits there, and read some descriptions of the Islands (actually, though modesty forbids my saying so, some of my favorite writings are my own), while listening to some authentic island music. It warms the cockles of my heart, though it gives me itchy feet.

That is what I did after my latest meeting with Bill, and as I was doing it I began to suffer pangs of conscience. It reminded me that the last time I was in the Islands, I didn't even mention it in these columns, and that will be two years ago come December. It isn't that I didn't revel in it, because I did. I think I may have kept mum because I felt sort of like a fellow does when he has stood up his best girl to go out with another one on the spur of the moment, and gotten caught at it. Not that what I did was on the spur of the moment. It was planned that way. In the first place, I have no pictures to show for the visit. For the first (and probably the last) time I took a video camera instead. The tape is great, but never where I want it, which is why I turned years ago from slides to prints. But that is not the real reason, or at least in thinking about it, I don't believe it is.

The real reason, I think, is that I had stood up my Special Island, my Bali H'ai, and gone a whoring after a false god(dess) named Maui, and was afraid that if I wrote about it, Kauai might find out and stop being my Special Island. Like most false gods (and goddesses), this one was very lovely. To many people who march to the island drummer, *Maui* is their Special Island. It was to Charles Lindbergh, who lived out his last days and was buried there. Maybe it is because I visited Kauai first that she has continued on as my Bali H'ai, but however that may be, Kauai has assured me she will forgive me and remain my Special Island even if I sing Maui's praises a little bit. So I will.

Now that several airlines offer direct service from the continent to Maui, bypassing the mess in Honolulu, it has become the place to go for meetings. In fact, I had scheduled a course there this month, but had to give it up because of other commitments. (I was going to stop by Kauai, too, thanks.) Maui's long west coast has wide sand beaches, for which the trade winds assure mostly sunny, warm weather year-round. Many large resort hotels have congregated there. The trade winds unload their moisture on the east coast, so that little is left by the time they cross the central mountains; it keeps the east side wet, lush, and green. This is true of all of the islands, though the amount of rain depends on land mass. Since Kauai's is small, it gets relatively less water on the coast. Most of it falls inland, on Mount Waialeale, with 500 inches a year the wettest place on earth, and on the Alakai Swamp atop the island's central core. The drive to Hana, on Maui's lush east coast, is one of the loveliest anywhere, ablaze as it is with the brilliant royal poinciana and other tropical blooming trees and plants. It is just below Hana that Lindbergh lived and is buried in a lovely little country churchyard. Not only was the area closed entirely to the public shortly after I was there (it is on private property), but I understand that now its presence on the island is not even mentioned anywhere, in an attempt to halt its desecration, indeed the ultimate destruction, by souvenir hunters.

Though the active firepit, Halemaumau, in Volcano National Park atop Kilauea on the Big Island of Hawaii, is better publicized and oftener visited, and though Mauna Loa, on the same island, has been responsible for more fireworks, nothing can touch the giant crater of Maui's Haleakalah as a sheer spectacle. To catch its full force, one needs to watch the sun rise over its rim, casting its early slanting red rays over the mist that generally fills much of the 3,000-foot deep, 3 by 2 mile cauldera, gradually spreading over the stark, variegated lava formations that form its walls. A shelter is provided against the cold, which is intense, much of it from a severe wind chill. As we started up the long slope in the pre-dawn darkness a light rain was falling, turning to snow at the 4,000-foot mark. We later broke out of the clouds, and on the 10,000-foot summit skies were clear. By the time we had reached the foot on our return, all was in bright sun; that is the time for one to fly into the cauldera in a helicopter.

Why, then, with all that glamour and marvelous scenery on Maui do I continue to turn to Kauai? There are more and better resorts on both the Big Island and Maui—or at least there have been. After all, it has been five years since I've been to Kauai, though it doesn't seem that long. But Kauai is quieter, and has areas more secluded, still. Nothing on any other island that I have seen approaches the grandeur of Waimea Canyon and the Na Pali Coast. The 4,000-foot high Alakai Swamp still beckons. Perhaps those things that I mentioned when last I wrote—which was 1981—are among the vast number of my things to do that I shall never do; some of those things, incidentally, are within my arm's reach right now. And there are so many other places to satisfy wanderlust, so many islands yet unvisited. That's why it was Maui last time, and not Kauai.

It's a good thing Kauai is not a jealous mistress. In dreams and reminiscences, as with Mark Twain a hundred years ago, Kauai still reigns supreme, and is atop my list of places to go back to. But as long as most of my travel time is medically determined (I mean through business and CME), she may have to wait a while longer. Then again, one never knows when the time will come to make my travels to other, more perfect worlds. The only thing predictable is that that time grows closer each year—in fact, each day and hour.

Aloha nui loa, O Kauai, ā hui hou kākou.

J.B.T.

in memoriam

Howard A. Farrar, age 75. Died November 6, 1986. Graduate of University of Tennessee College of Medicine. Member of Coffee County Medical Society.

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A Case of Wound Botulism

BOBBY C. HIGGS, M.D.

Introduction

From the reports in the literature, wound botulism is extremely rare; in an article in 1973, Merson and Dowell record only four cases reported in the United States prior to 1971. From the Centers of Disease Control in Atlanta another five cases were reported over the next one and a half years.[1] In 1982 an article by Keller et al[2] cites only 21 cases reported. At any rate, the numbers are few, and according to the Tennessee Department of Health and Environment, this case may be the first reported in our state,[3] and perhaps one of the first from the South.[1]

A review of this case and brief discussion of wound botulism may serve to alert the Tennessee physician to the seriousness and rapid development of symptoms encountered by all practitioners.

Case Report

A 13-year-old boy came to the office on May 23, 1985, complaining of weakness and dizziness, difficulty with vision, specifically double vision, and difficulty swallowing. His voice was becoming weaker, and speaking was increasingly difficult. Twenty-four hours earlier he had headaches and according to the informant he was sleeping more than normal. His temperature was not taken, but he "felt warm" according to his parents. Because of these symptoms, he was admitted to the hospital for further evaluation and observation.

Approximately eight days earlier, the patient had sustained a significant crush injury to the right foot while riding on the back of a piece of farm machinery. The injury required 12 sutures after thorough irrigation and debridement in the Jackson-Madison County General Hospital emergency room; tetanus toxoid was administered. On the day of admission to the hospital, he was reexamined by the attending surgeon and approximately 100 cc of serosanguineous blood was removed from the injury site; it was sent to the laboratory for culture and sensitivity studies.

On admission the white blood cell count was 20,900/cu mm, with 89% segmented neutrophils, 1% band forms, and 10% lymphocytes. Platelets were normal, and urinalysis was normal with a specific gravity of 1.030, a pH of 6, and 0 to 3 white cells per high power field; sugar, protein, blood and ketones were negative.

Approximately four hours after admission, the patient's condition had deteriorated significantly. Cranial nerve abnormalities were increasing quite rapidly, with bilateral ptosis, weakness of extraocular movements, slurred and difficult speech, weak voice, double vision, significant swallowing impairment with thick mouth secretions and a thick tongue, and there was obvious extremity muscle weakness. At this time, the diagnosis of wound botulism was entertained. No botulinum antitoxin was readily available, as its only source is the CDC in Atlanta. After consultation with LeBonheur Children's Hospital in Memphis, the patient was transferred there immediately by ambulance; the CDC released, by air shipment, the necessary polyvalent botulinal antitoxin.

Respiratory arrest subsequently followed requiring ventilatory assistance for approximately seven days. On May 24, 1985, surgical debridement was carried out on the injured foot, and a pigskin covering was placed over the injury site by the plastic surgeon. On May 30, the plastic surgery department did an extensive procedure, with debridement of the right foot, heel, and ankle, and reconstruction of the heel and foot area were performed using a latissimus dorsi microvascular flap and a split thickness skin graft.

Samples of serum, stool, and wound exudate were sent to the Nashville Reference Laboratory of the Tennessee Department of Health and Environment, where *Clostridium botulinum* type B toxin was found by mouse assay and culture from the exudate samples; toxin assays from the serum and stool were negative. The serosanguineous material aspirated from the foot on the day of admission to our hospital

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From the Department of Pediatrics, Jackson Clinic, Jackson.
Reprint requests to 616 W. Forest, Jackson, TN 38301 (Dr. Higgs).

was reported as positive for *Enterobacter cloacae*, alpha-hemolytic *Streptococcus*, and *Clostridium perfringens*.

The patient was discharged from the hospital in Memphis on July 3, 1985, and has been seen in our office on one occasion, on July 20, 1985, with an upper respiratory infection. In spite of the seriousness and rarity of this disease and the extensive treatment that he required, he has shown an excellent recovery.

Comment

Wound botulism was first reported in the United States in 1951, although the reported case occurred in 1943.[4] The case reports now total 31, with our case being the first reported in the nation for 1985. Little is written in the literature concerning wound botulism, although clinically botulism is very well defined. Four types of botulism are now recognized: food-borne, infant, wound, and unclassified.

Food-borne botulism is the best known and the most common type. Numerous outbreaks have been recorded in the past, and symptoms occur within hours of toxin ingestion.[5] Within 12 to 36 hours gastrointestinal symptoms of nausea, vomiting, abdominal cramps, and distention appear. Constipation is a common complaint, and a few will experience diarrhea. From hours to days later, systemic neurologic symptoms develop; as they closely resemble symptoms of other types of botulism, they will be discussed under wound botulism.

Food-borne botulism often involves more than one individual, occurring in outbreaks in which the involved persons have shared a common food. Apparently the sicker persons with the poorer prognosis will be the first to present themselves to the physician, and will have ingested the greater amount of toxin.[6] A high index of suspicion is important in making the diagnosis.

Infant botulism was first reported in 1976.[6] Characteristically, infants from 1 to 6 months old are involved, and symptoms are most often feeding difficulty, weakness, constipation, and lethargy that develop insidiously. I have had one case of infant botulism in which the diagnosis was suspected because of the regression of developmental milestones. Extreme muscular weakness, refusal to feed, and constipation were the presenting symptoms of this 16-week-old infant.

Laboratory data suggest infant botulism is caused by the toxin produced in the gut by the ingested organisms, rather than through the ingestion of preformed toxin. For some time now, the American Academy of Pediatrics has advised against the use of honey for infants because of the possible contamination with *C. botulinum* organisms.

Respiratory insufficiency and arrest may occur in infant botulism, and the diagnosis requires close observation of the infants. It is of interest that some infants dying of SIDS have had *C. botulinum* cultured from the stool specimen.[7] The peak incidence of both SIDS and infant botulism closely parallel one another with regards to age of occurrence.

Wound botulism is an extremely serious illness that can result in death if not diagnosed and treated appropriately. Due to the few clinical reports in our literature, it is not often considered in the differential diagnosis. *C. botulinum* is an anaerobic spore-forming Gram-positive rod found commonly in the soil and water, and consequently it would be expected to be a common contaminant of many wounds.[8]

Typically, wound botulism produces a bulbar paralysis and descending symmetrical motor paralysis. The highest cranial nerves are affected first, and diplopia and ophthalmoplegia may be the first symptoms. Following this may be blurred vision, dry mouth, dysarthria, dysphagia, abdominal symptoms, and ultimately respiratory difficulty with failure.[8]

Differential Diagnosis

It is of the utmost importance in the treatment of wound botulism to consider it in the differential diagnosis, which also includes the various neuromuscular pathological processes, among them Guillain-Barré syndrome, diphtheria, myasthenia gravis, tick paralysis, and organophosphate insecticide poisoning. Each has its distinctive clinical features and must be considered in a case such as this.

Guillain-Barré syndrome is differentiated by an ascending motor paralysis, abnormal spinal fluid findings, parasthesias, and infrequent cranial nerve involvement. Myasthenia gravis is characterized by an abnormal fatigability of the skeletal muscle and responds quickly and dramatically to neostigmine.

Two not uncommon problems in pediatrics are tick paralysis and organophosphate poisoning. Tick paralysis is also characteristically an ascending flaccid motor paralysis, which rapidly responds to the removal of the offending tick. Organophosphate poisoning causes a more generalized effect by inhibiting the enzyme cholinesterase and producing an accumulation of acetylcholine. Nicotinic effects produced are generalized muscle weakness, easy fatigability, muscle twitchings, tremors, and cramps. There are no characteristic changes in tendon reflexes, which may be hypoactive, hyperactive, or normal.

Muscarinic effects are nausea, vomiting, abdominal cramps, sweating and extensive lacrimation, salivation, dyspnea, and profuse bronchial secretions. Atropine produces dramatic and rapid improvement in this condition.

Any disease or injury that is capable of affecting the central nervous system must be included in the differential diagnosis, and the textbooks may be consulted for a more complete listing.

Treatment

Immediate attention to treatment is necessary for survival. From the previously reported cases, the role of antibiotic therapy is rather unclear. It did not prevent the development of wound botulism, but Kelley[7] recommends antimicrobial therapy in all patients because of the opportunity it provides to eliminate *C. botulinum* organisms from the gut or wound, which cannot be eliminated by other means. Pencillin derivatives are preferred.

Guanidine hydrochloride therapy is mentioned in several of the reports but its use is not demonstrated to be efficacious. Aminoglycosides should not be used because of the recent reports with infant botulism suggesting that they worsen the neuromuscular weakness.[2,7]

For the most part, supportive care is the most important aspect of treatment for a successful outcome. Monitoring of vital signs will dictate the need for respiratory assistance.[7] Specific antibotulinum therapy is indicated in symptomatic patients; the botulinum antitoxin can be obtained from the CDC in Atlanta.

Our patient had a good recovery, although a long and involved hospital stay with surgery and ventilatory assistance was necessary. His is an interesting and unusual case, perhaps the first reported case of botulism in the state of Tennessee, and we present it simply to increase the awareness of the disease diagnosis.

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APRIL 1987

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
			1	2	3	4
5	6	7	8 TMA 152ND ANNUAL MEETING Hyatt Regency Hotel—Knoxville	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30		

Suppurative Aortitis

GEORGE W. HOLCOMB, III, M.D. and BRUCE M. SMITH, M.D.

The spectrum of arterial infection includes endarteritis, the colonization and infection of aortic aneurysms, invasive arterial wall infections, and the infections of prosthetic grafts. In the absence of prosthetic material, preexisting mural damage at the time of bacterial exposure appears necessary for the establishment of clinically important arterial infections, and these resultant transmural infections of the arterial wall only rarely have been recognized in time for successful management. We describe four cases of suppurative aortitis treated at Vanderbilt University Medical Center, review nine previously reported cases, and propose guidelines for the treatment of these infections.

Case Reports

Patient 1. An anephric 52-year-old white man who had been receiving hemodialysis three times a week for the past 14 years underwent bilateral nephrectomy in 1969 for end-stage renal disease of unknown etiology; four subsequent renal homografts failed. Chronic osteomyelitis of his left hip had been treated with weekly intramuscular injections of vancomycin for the three years prior to his admission. He came to the Nashville Veterans Administration Medical Center in January 1983 complaining of severe abdominal and back pain, chills, and malaise, and had vomited on several occasions in the 24 hours prior to his admission.

When seen initially, he was observed to be weak and rigorous, with darkly pigmented skin. His temperature was 96.6°F, his pulse was 96/min, blood pressure 132/90 mm Hg, and respiratory rate 16/min. Cardiac examination was normal. His abdomen was nontender, slightly distended and tympanitic, no abdominal masses were palpable, bowel sounds were active, and abdominal bruits were absent. An uninflamed external dialysis shunt was noted in his left leg. Pain limited the movement of his left hip. The admission hematocrit was 28% and the leukocyte count was 5,500/cu mm, with 78% segmented neutrophils, 15% lymphocytes and 5% monocytes. Serum electrolytes were normal, blood urea nitrogen was 100 mg/dl, and creatinine 10.3 mg/dl. Marked calcification in his thoracic and abdominal aorta and in abdominal aortic branches was noted on chest and abdominal radiographs (Fig. 1). On the third hospital day his fever rose to 103°F following hemodialysis, and treatment with tobramycin intravenous (IV) was begun; febrile episodes continued over the next five days. Blood cultures grew *Escherichia coli*, sensitive to tobramycin. A radionuclide biliary scan and abdominal ultrasound were normal. On the 11th hospital day he underwent abdominal computed tomographic (CT) scan, which demonstrated a periaortic mass immediately below the diaphragmatic hiatus (Fig. 2). His hematocrit had fallen to 21% from 25% two days previously.

Emergency abdominal aortography demonstrated diffuse atherosclerotic disease with deformity of the right posterolateral surface of the aorta (Fig. 3). Exploratory laparotomy through a left thoracoabdominal incision exposed a periaortic abscess and hematoma cephalad to the celiac trunk adjacent to a 1.5-cm diverticulum of the posterior aortic wall. There was no evidence of circumferential aneurysmal dilatation, and no active bleeding was seen. Aerobic and anaerobic cultures were taken and Gram stain of the purulent material demonstrated Gram-negative rods. The abdomen and chest were temporarily closed, and a left axillo-bifemoral bypass was performed with a 6-mm expended polytetrafluoroethylene (PTFE) graft. Following completion of this procedure, the aorta was reexposed and clamped above and below the diverticulum. The aorta was opened and demonstrated plaquing and irregularity on its posterior surface at what was presumed to be the site of leakage. This segment of the aorta, which was extremely friable, was excised and sent for histologic examination and culture. An excellent pulse in the distal aortic stump was transmitted retrograde from the axillo-bifemoral bypass. The proximal and distal aorta were oversewn and a silastic suction catheter was placed within the abscess cavity.

Postoperatively, the periaortic collection and aortic wall cultures grew *E. coli* and the patient was treated with clindamycin and tobramycin. Histologic sections showed transmural inflammation and hemorrhage. Because of persistent acidosis and suspected bowel ischemia the patient was returned to the operating room the first postoperative day, but his bowel was normal, and it appeared that the acidosis was the result of continued sepsis and marginal perfusion of his lower extremities. A supplemental right axillo-femoral bypass with PTFE was performed, and a peritoneal dialysis catheter was inserted because his dialysis shunt had clotted. His condition did not improve, and after becoming increasingly acidotic and hypotensive, he died on the fifth postoperative day. Postmortem examination was not allowed.

Comment: The patient's poor peripheral perfusion presumably was based on continued sepsis, since core perfusion, as indicated by viable bowel, appeared adequate. A 6-mm graft was chosen because of the patient's small arteries. We cannot determine whether the involved area of aorta would have produced free rupture or whether an in-situ graft would have altered the patient's course.

Patient 2. A 77-year-old white man had undergone left hemiglossectomy and radical neck dissection in 1957 for squamous cell carcinoma of the floor of the mouth. Although he had cervical nodal metastases at that time, he was without clinical recurrence when admitted to Vanderbilt Hospital complaining of abdominal pain, constipation, and intermittent diarrhea for three weeks in December 1964. He had noted increasing abdominal distention for the past week. When seen initially, his temperature was 98.6°F, his pulse was 84/min, blood pressure 210/90 mm Hg, and respiratory rate 20/

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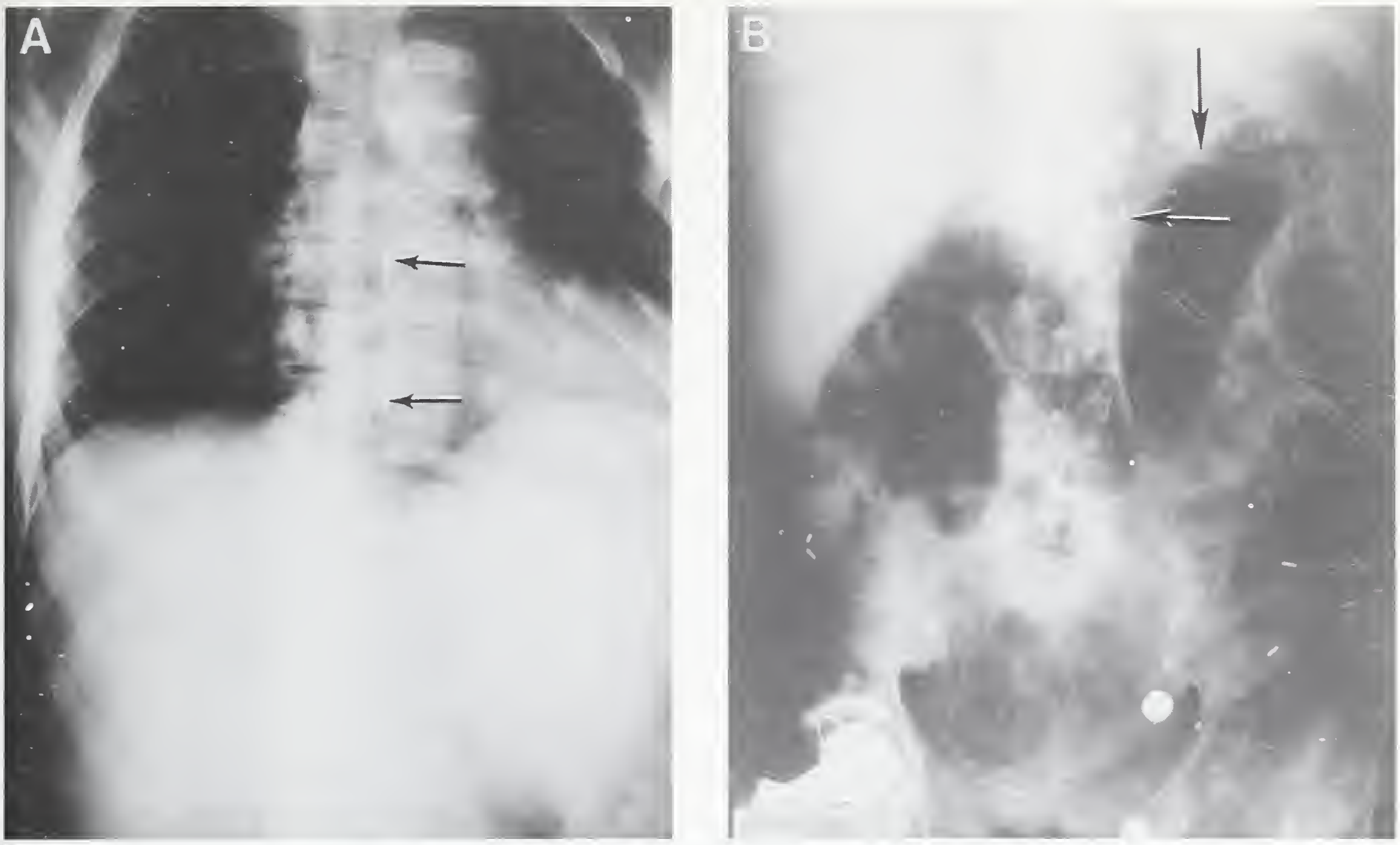

Figure 1. (A) Chest radiograph in patient 1 demonstrates calcification (arrows) and tortuosity of the thoracic and abdominal aorta. (B) Supine abdominal radiograph demonstrates marked calcification of the aorta and visceral vessels (arrows).

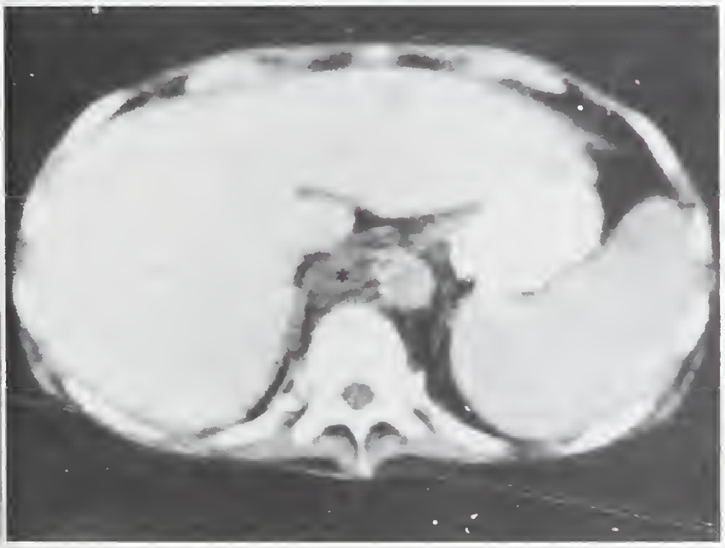

Figure 2. Abdominal CT scan in patient 1 demonstrating a large periaortic mass (asterisk) just below the esophageal hiatus. No evidence of aneurysmal disease is seen in the aorta.

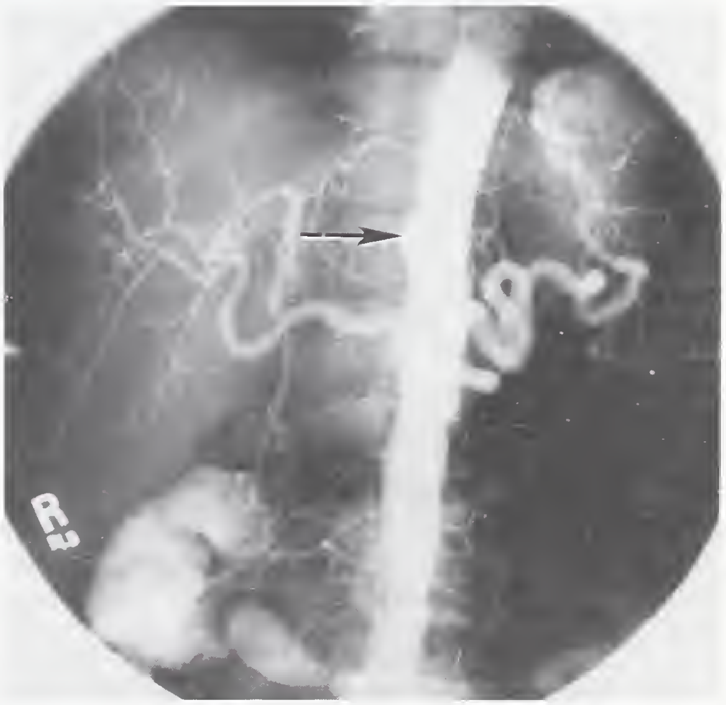

Figure 3. Abdominal aortogram in patient 1 at the level of the celiac axis and superior mesenteric artery demonstrates an irregularity of the right posterolateral wall of the aorta superior to the origin of the celiac axis (arrow).

min, and there was no evidence of tumor. Cardiac examination was normal, and his lungs were clear. His abdomen was soft but distended, with a few high-pitched bowel sounds. No organomegaly or masses were present.

Admission hematocrit was 45%, and his leukocyte count was 17,000/cu mm, with 85% segmented neutrophils. Serum electrolytes were normal, BUN was 67 mg/dl, and urinalysis was unremarkable. An electrocardiogram suggested left ventricular hypertrophy. A chest radiograph was normal, but cholelithiasis was present on abdominal radiographs, and on upper gastrointestinal (GI) series a 6-cm stricture suggestive of carcinoma was noted in the third portion of the duodenum. After this condition deteriorated markedly, with protracted vomiting, increasing abdominal distention, and a falling hematocrit, and he died shortly thereafter. Postmortem examination revealed 2 liters of blood in the abdominal cavity; the anterior aortic wall 2 cm below the renal arteries was necrotic, and an extensive hematoma encircled the third portion of the duodenum. There was no evidence of duodenal tumor. Histologic examination demonstrated moderate atherosclerotic changes in the aorta, and acute and chronic inflammatory reaction in the aortic wall (Fig. 4). Gram stains demonstrated Gram-positive cocci in the aortic wall. Postmortem cultures grew enterococcus, and acute bronchitis was also discovered with similar organisms on Gram stains and culture. There was no evidence of recurrent carcinoma.

Comment: Retroperitoneal hematoma mimicked duodenal carcinoma in this patient. The presumed source of aortic infection was the tracheobronchial tree.

Patient 3. A 23-year-old white woman with a known patent ductus arteriosus was admitted to Vanderbilt Hospital in November 1973 complaining of cough, headaches, coryza, and fever one week after receiving a flu shot. Her temperature was 104°F, her pulse was 150/min, respiratory rate 30/min, and blood pressure 200/70 mm Hg. A sinus tachycardia was present with a grade 4/6 machinery murmur heard in the left second and third intercostal spaces. A grade 2/6 systolic murmur was heard over the precordium, radiating into the carotid arteries. Femoral and pedal pulses were diminished and delayed relative to the radial pulses.

The admission hematocrit was 30.4%. A leukocyte count was 10,500/cu mm, with 76% segmented and 16% juvenile neutrophils. Serum electrolytes were normal and the urine contained 8 to 10 white blood cells and 3+ bacteria per high powered field. Chest radiographs and electrocardiogram demonstrated left ventricular hypertrophy. Blood and urine cultures grew a coagulase negative *Staphylococcus* and subacute bacterial endocarditis was diagnosed. Intravenous cefazolin was administered and her clinical condition improved.

On her tenth hospital day, cardiac catheterization and aortography demonstrated a patent ductus arteriosus and postductal coarctation of the aorta. There was no evidence of aortic aneurysmal dilatation. On her 17th hospital day she began to vomit bright red blood and suffered a cardiac arrest. After she was resuscitated she was taken immediately to the operating room, where through a left thoracotomy the posteromedial portion of her descending aorta was found replaced by a large false aneurysm extending into her mediastinum and esophagus. After securing proximal and distal control of her aorta, the ductus was divided and the pulmonary arterial side was ligated. After the diseased aorta was debrided, a 20-mm woven dacron prosthesis was implanted. Histologic sections of the resected tissue showed marked acute and chronic inflammatory changes. No organisms were seen on histologic sections of her aorta, but culture of aortic wall grew coagulase negative *Staphylococcus*.

She was discharged six weeks after her operation after a smooth postoperative period. She had received six weeks of IV cefazolin, and four blood cultures were negative on discharge. Thirteen years postoperatively she is alive and well, and is being followed at six-month intervals.

Comment: In selected circumstances, in the absence of a preexisting prosthesis or gross purulence, in-situ reconstruction of the infected aorta appears to be appropriate. A prosthetic graft was, in fact, the only practical option available for this patient because of the extensive aortic defect and the need to debride the grossly abnormal aortic wall. The presumed site of infection was thought to be the urinary tract.

Patient 4. Eight days after admission to the Middle Tennessee Tuberculosis Hospital in 1969 for miliary tuberculosis, this 68-year-old man began to have hematemesis and melena requiring nine units of blood. At emergency laparotomy a 10-cm unruptured abdominal aortic aneurysm was found to have eroded into the posterior wall of the duodenum. Because the hospital did not have the facilities for definitive repair, following control of hemorrhage and closure of the duodenum, the patient was transferred to Vanderbilt Hospital still under anesthesia.

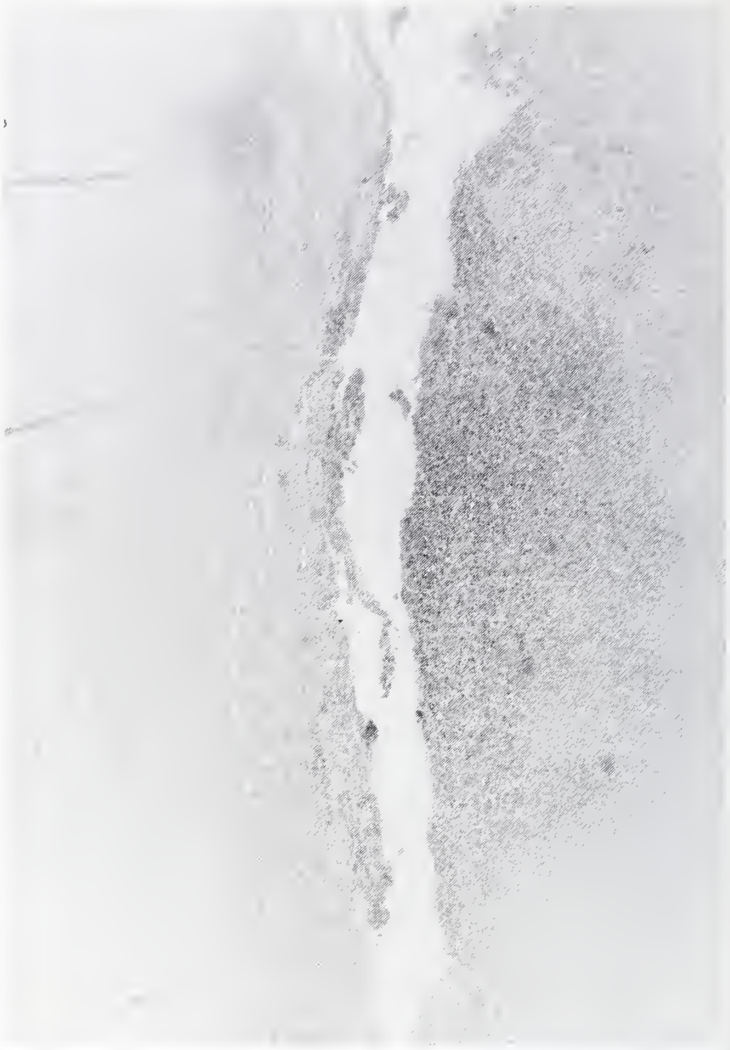

Figure 4. Histologic examination of the aortic wall in patient 2 demonstrates moderate atherosclerosis without aneurysm formation. Acute and chronic inflammatory reaction extends along the periadventitial layer of the aorta and into the adjacent connective tissue (X100).

Upon arrival, his blood pressure was 130/70 mm Hg and pulse 104/min. He was taken to the operating room where the aneurysm was noted to originate 1 cm below the renal arteries and to involve the iliac vessels. The diseased aorta and iliac arteries were excised and replaced by a dacron bi-iliac graft. The previous closure of the duodenum was reinforced with a layer of 4-0 silk Lembert sutures.

His postoperative course was complicated by high output renal failure, and he required chronic ventilatory assistance. Twelve days postoperatively necrosis of his left colon necessitated colectomy and transverse colostomy. He remained unresponsive postoperatively, and his condition gradually deteriorated, with *E. coli* septicemia, anemia, and hypotension. He died on the 20th hospital day.

Cultures of his sputum subsequently were positive for *Mycobacterium tuberculosis,* and histologic examination of his abdominal aorta demonstrated extensive caseous necrosis of the adventitia and media. Many granulomata with acid-fast organisms were found along with extensive endarteritis obliterans of the vasa vasorum. There was moderate atherosclerosis present.

Comment: In spite of the current controversy and good results with in-situ grafting of spontaneous atherosclerotic aorto-duodenal fistulae, in retrospect, placement of an extracorporeal bypass and suspecting and treating the tuberculous aortitis with specific antimicrobial agents might have altered the patient's course.

Collected Series

Thirteen patients, including those in our series, have been documented as having invasive infections of the aorta[1-7] (Table 1). Seven have been male and six female, with a mean age of 62.6 years (range 23 to 77 years). Ten of the 13 patients were 60 years old or older. Six of the patients (46%) died prior to operation, three (23%) died postoperatively, and four (31%) have survived 9 months to 12 years; one surviving patient died 20 months postoperatively of carcinoma of the lung. Of the nine previously reported cases, eight aortic cultures (89%) grew *Salmonella,* suggesting seeding from the gastrointestinal tract. In contrast, a variety of sources are implicated by the spectrum of organisms found in our series.

Discussion

Intact endothelium is extremely resistant to colonization or infection during periods of bacteremia from either naturally acquired or iatrogenic infections.[1,8-12] The development of invasive intramural arterial infection following bacteremia, therefore, appears to require facultative injury to the endothelium or vessel wall from atherosclerosis, accidental or surgical trauma, or congenital defects.[12] Each of the patients in our series had such an abnormality, which provided a fertile culture medium for invasive infection following bacteremia.

The damaged artery may become infected through a variety of routes. In addition to bacteremic seeding of plaques and aneurysms (probably the most important mechanism of end-

TABLE 1

COLLECTED SERIES: SUPPURATIVE AORTIC INFECTIONS

Patient	Age/Sex	Site	Organism	Pathology	Operation	Outcome (follow-up)
Vanderbilt Series						
No. 1	52/M	Supra-celiac aorta	*Escherichia coli*	Atherosclerotic aorta with inflammation and periaortic abscess	Debridement, axillo-bifemoral bypass	Died
No. 2	77/M	Infrarenal aorta	Enterococcus	Atherosclerotic aorta with acute and chronic inflammatory changes	None	Died
No. 3	23/F	Patent ductus arteriosus	Coag. neg. *Staphylococcus*	Acute and chronic inflammation	Debridement, in-situ bypass	Survived (living after 13 years)
No. 4	68/M	Infrarenal aorta	*Mycobacterium tuberculosis, E. coli*	Caseous necrosis	Debridement, in-situ aorto-bi-iliac graft	Died
Collected Series						
No. 5[1]	67/F	Infrarenal aorta	*Salmonella typhimurium*	Suppurative aortitis	Emergency laparotomy	Died
No. 6[2]	60/F	Infrarenal aorta	*S. cholerasuis*	Suppurative aortitis	None	Died
No. 7[3]	74/M	Infrarenal aorta	*S. newport*	Lumbar osteomyelitis with extension into aorta	None	Died
No. 8[3]	75/F	Infrarenal aorta	*S. typhimurium*	"Ruptured aorta," colitis	None	Died
No. 9[4]	67/F	Infrarenal aorta	*S. typhimurium*	Aortic wall abscess with rupture	None	Died
No. 10[5]	52/M	Infrarenal aorta	*S. cholerasuis*	Suppurative aortitis with rupture	Debridement, in-situ bypass	Survived (living after 4 months)
No. 11[6]	73/M	Infrarenal aorta	*Arizona hinshawii*	Microabscesses in aortic wall	Debridement, extra-anatomic bypass	Survived (living after 9 months)
No. 12[7]	65/F	Infrarenal aorta	Beta-hemolytic *Streptococcus*	Septic necrosis of aortic wall	Emergency laparotomy	Died
No. 13[7]	61/M	Infrarenal aorta	*S. enteritidis*	Aortitis with transmural organisms	Debridement without revascularization	Survived (living after 20 months)

arterial infection) invasive intramural infection of the arterial wall may also occur from bacteria-laden emboli or from endothelial vegetations which impinge upon, damage, and subsequently invade the normal arterial wall.[13,14] Additionally, *syphilitic aortitis* is a rare but classic example of vessel wall invasion via the vasa vasorum causing inflammatory destruction of the media.[12] While more common pathogens might theoretically invade the arterial wall via this route (patient 4), it has not been possible to document this as an important mechanism clinically. Arterial infections may also occur as an extension of a *contiguous infectious process,* either directly or through perivascular lymphatics or adjacent lymph nodes,[15] and *intra-arterial injection of illicit drugs* likewise may result in the infection of a pseudoaneurysm.

The presence or absence of sepsis that might indicate invasive infection should determine the management of the patient in whom aortic infection is suspected. Colonization of aortic endothelium without mural invasion is usually suspected during elective aneurysmectomy, and standard prosthetic grafting appears to be acceptable management.[7] Radically different management is indicated, however, in the presence of transmural infection suggested by fever, leukocytosis, or positive blood cultures, or where periarterial suppuration is encountered at operation. These patients should be managed by debridement of all infected tissue and extra-anatomic bypass. In contrast to the asymptomatic group, these patients have an extremely poor prognosis.[16,17]

The *diagnosis* of aortic infection is often difficult, and delay in diagnosis contributed to the unsatisfactory results in our series. An atherosclerotic abdominal aorta is only rarely considered a source of positive blood cultures, but fever in the presence of an aneurysm should raise the index of suspicion of true arterial infection and suggest the possible need for an unconventional operative procedure.[17] Jarrett et al[18] noted that early diagnosis and intervention played a major part in survival of their patients with infected abdominal aneurysms. Six of seven patients in whom the diagnosis of infected aortic aneurysm was made on admission survived, whereas only one patient survived among nine admitted with the diagnosis unclear; three of those died without the correct diagnosis having been made.

Preoperative diagnostic studies such as aortography may document aneurysm formation, but rarely give any indication of potential arterial infection. Cooke and Ehrenfeld[19] have described an infected "unusual, lobulated, saccular aneurysm," clearly different from the usual atherosclerotic aneurysm. Miller et al[20] have described a similar appearance of the aorta in an aneurysm infected with *Histoplasma capsulatum.* In the febrile patient this radiographic picture may be helpful in the diagnosis of suppurative aortitis. CT scanning of the abdominal aorta may demonstrate small pockets of gas in infected clots around the prosthetic grafts, which can be differentiated from those normally found within bowel.[21] Radionuclide-labeled leukocytes have also been shown to outline paraprosthetic aortic graft infections; this might logically be extended to suppurative aortitis or infected aneurysms.[22]

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Special Communication

Nine-Year Cough Aborted by TENS

CARL ROGERS, M.D.

All 17 of her physicians, including several Nashville specialists and those involved in a four-day workup at the Mayo Clinic, agree that her symptom must surely be on an allergic basis. This opinion prevails in spite of a completely negative allergy workup, including the routine allergens, RAST testing, and Mecholyl levels for asthma, plus an otherwise negative allergy history. Not a single skin test showed the least bit of reaction.

She is a 34-year-old housewife, mother, and schoolteacher. For the past nine years she has developed a cough on October 1. It lasts until February 15, when it clears as rapidly as it started. The other eight and one-half months are free of cough.

She has two or three days of progressive chest tightness preceding its onset enabling her to predict that the cough is on its way. The cough is nonproductive and very persistent, lasting 24 hours a day for three and a half months. It starts at the upper tracheal level. There is no increase in nasal or postnasal drainage and there is no conjunctivitis or tearing. There is no wheezing, but later in the day she may have a tightness in the chest. It is aggravated by prolonged talking and by lying down. She has to sleep in a recliner. It is also aggravated by an environment that is too hot for her. She has to do a lot of her teaching standing in the open outside door of her classroom.

She has had repeated x-rays, laryngoscopy, bronchoscopy, two CT scans, thyroid studies, a GI series with barium swallows, EKGs, and cardiac studies, in addition to allergy workups. Psychosomatic and biofeedback studies have not been done.

She has been treated with numerous antibiotics, including sulfa drugs, even though she does not have fever and chills. She has taken practically every antihistamine preparation and antitussive, including codeine and hydrocodone. Intal, Vancenase, Proventil, Nasalide and Azmacort inhalers have not provided relief, even with prolonged use.

This year she had a fair remission for about seven days while on a ten-day course of prednisone in a dosage of 10 mg four times a day. This had to be discontinued because of severe weakness, anorexia, nausea and vomiting, facial edema, and emotional and mild mental changes. This response to large doses of steroid did, however, serve to strengthen the belief that an allergy was involved.

Two years ago she was put on a saturated solution of potassium iodide on the chance that she just might have thyroid pressure on the trachea; this gave no relief. A course of indomethacin (Indocin) had to be discontinued because of vomiting.

So much for what has been done during the past nine years.

One week ago as I watched the nurse place a transcutaneous electronic neural stimulation (TENS) unit on an abdominal wound of another patient, it suddenly came to mind that if this unit can inhibit or block the pain impulses from reaching the brain, perhaps it could do the same for cough impulses. So a TENS unit and electrodes were applied to the cough patient. Instructions with the unit caution against applying the electrodes over the neck because of the possibility of pharyngeal and laryngeal spasm, but since her cough starts just below the larynx the electrodes were applied over that area. She was monitored closely as the intensity was increased, but she had no trouble. She was cautioned to be aware of the possibilities, and was instructed as to what to do should any swallowing or breathing problems develop.

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The TENS unit should not be used over the carotid sinus or in patients with demand pacemakers. Other contraindications are not mentioned in the available literature.

For the first few hours the patient could not tell much difference, but after five or six hours she noticed that the urge to cough was subsiding a great deal. For the first time (except when on the high dosage of steroid) she was able to sleep all night lying flat without the cough. The next day three different fellow teachers asked, "What has happened to your cough?"

For the past week she has been relatively free of cough with the unit on fairly low voltage. While changing the electrodes the cough starts up again but in two or three minutes after new electrodes the cough disappears. She now sleeps lying flat, talks as much as she desires, and tolerates heat better. The nausea has cleared up, probably due to no medication as much as anything. Her mood is better and she is functioning normally again.

According to the manufacturers there are no ill effects from prolonged usage of TENS for pain, so presumably there would also be none for cough. She is being monitored closely for any possible ill effects.

Certainly a series of cases will be needed before the TENS unit can be added to the armamentarium for cough control. It may be found useful in controlling persistent coughs in lung conditions such as neoplastic disease, where limiting the cough would not have any ill effects.

The purpose of this report at this time is to suggest this possible use for the TENS unit to other physicians and therapists where cough control is difficult.

HELP FOR IMPAIRED PHYSICIANS

Through its Committee on Impaired Physicians, TMA helps doctors who are suffering from alcoholism and drug addiction. The thrust of the program is rehabilitative, not punitive. The Committee is composed of physicians who have special expertise in these areas, some from personal experience. Effective treatment for these illnesses is achieved most easily when the disease is detected early and family, friends, and associates are urged to avoid misguided sympathy which enables the condition to deteriorate.

HELP US TO HELP

Call the TMA Impaired Physician Program (615) 327-2711; outside Nashville call collect. Phone service available around the clock.

AMA 1986 Interim Meeting Report

The Fifth Pathway

Background

The Council issued its first policy statement on the Fifth Pathway in 1971. Modifications of the statement were made in 1973, 1975, and 1979. The 1979 policy statement, which is quoted below, describes the five possible routes for foreign medical students and graduates who wish to enter the American graduate medical education system, and sets forth the requirements for a Fifth Pathway program.

A. The Entry Routes

1. Admission with advanced standing to American medical schools.
2. Certification by the Educational Commission for Foreign Medical Graduates (ECFMG) on the basis of satisfying the ECFMG educational requirements, as well as passing the ECFMG examination.
3. Obtaining a full and unrestricted license to practice medicine, issued by a state or other U.S. jurisdiction authorized to license physicians.
4. In the case of U.S. citizens, successfully passing the complete licensure examination in any state or other licensing jurisdiction in which the law or regulations provide that a full and unrestricted license to practice medicine in that state or jurisdiction will be issued to the physician after satisfactory completion of his internship or residency in that state, without further examination. To be eligible for this route, the foreign medical graduate must have completed all educational requirements that would make him eligible for ECFMG certification should he choose to apply.
5. Completion of the "Fifth Pathway" as described in the statement below.

B. The Fifth Pathway Statement

1. A new pathway for entrance to approved programs of graduate medical education, other than those existing under previous policies, became available as of July 1, 1971, for students who have fulfilled the following conditions: (a) have completed, in an accredited American college or university, undergraduate premedical work of the quality acceptable for matriculation in an accredited U.S. medical school; (b) have studied medicine at a medical school located outside the United States, Puerto Rico, and Canada, which is listed in the *World Directory of Medical Schools*, published by the World Health Organization; and (c) have completed all of the formal requirements of the foreign medical school except internship and/or social service. Those who have completed all of these requirements including internship and/or social service are not eligible.
2. Students who have completed the academic curriculum in residence in a foreign medical school and who have fulfilled the above conditions may be offered the opportunity to substitute for an internship required by a foreign medical school, an academic year of supervised clinical training (such as a clinical clerkship or junior internship) prior to entrance into the first year of approved graduate medical education. The supervised clinical training must be under the direction of a medical school approved by the Liaison Committee on Medical Education.
3. Before beginning the supervised clinical training, said students must have their academic records reviewed and approved by the medical schools supervising their clinical training and must attain a score satisfactory to the sponsoring medical school on a screening examination or procedure acceptable to the Council on Medical Education. The Council on Medical Education will consider the acceptability of any screening examination or procedure proposed by a sponsoring medical school.
4. Said students who are judged by the sponsoring medical schools to have completed successfully the supervised clinical training are eligible to enter the first year of approved graduate training programs without completing social service obligations required by the foreign country or obtaining ECFMG certification.
5. The Council on Medical Education has recommended to all state boards of medical examiners that they consider for licensure all candidates who have completed successfully the supervised clinical training on the same basis as they now consider foreign medical candidates who have received ECFMG certification.

Enrollments of Fifth Pathway Programs

Data drawn from the AMA Masterfile have shown that over 90% of licensed physicians with Fifth Pathway training received their medical educations in Mexico. Medical schools in Italy, the Philippine Islands, Belgium, and the Dominican Republic among others

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This is AMA Council in Medical Education Report C, submitted to the House of Delegates at its interim meeting in December 1986. Past House Action: I-84:193-210; A-82:130-133; A-80:113-122.

have contributed small percentages of Fifth Pathway graduates.

Nineteen medical schools responded affirmatively to the Liaison Committee on Medical Education (LCME) Annual Survey (1986-87) question on sponsorship of a Fifth Pathway program. In 1981-82, 25 medical schools stated that they would accept students.

Records maintained by the AMA Office of Physician Credentials and Qualifications listed 17 active programs in 1985-86 in Puerto Rico and 10 states—Arizona, California, Colorado, Illinois, Maryland, New Jersey, New York, Rhode Island, Tennessee, and Utah.

In preparation for this report a telephone survey of the 17 active programs was conducted on Aug. 1, 1986. Three of these programs have recently been discontinued; one will conclude at the end of academic year 1986-87; one will conclude at the end of academic year 1987-88; and another is considering discontinuation at the end of academic year 1986-87.

The 14 programs active in 1986-87 report an expected enrollment of 138 students. This number is subject to change; an accurate enrollment figure will be available in 1987 when the LCME Annual Survey for 1987-88 is completed.

Enrollments in individual programs vary from 0 to 30. In some states the program is mandated by law. Although such a state program may not have attracted applicants or found qualified applicants, it is still maintained on the AMA active program list.

Table 1 presents information drawn from LCME Annual Surveys on the activity of Fifth Pathway programs over 13 academic years from 1973-74 to 1985-86. Although some fluctations are apparent, activity built quite steadily in terms of total applications, entrants, and graduates from 1973-74 to 1981-82. Thereafter, there is an overall decline and a sharp diminution in all recorded measurements in 1985-86. If an enrollment of 138 for 1986-87 is sustained, academic year 1985-86 will mark the beginning of a significant contraction of the Fifth Pathway.

The Council attributes the decline in Fifth Pathway enrollments to several factors: (1) reported declines in enrollments of U.S. citizens in Mexican medical schools, and, in particular, the Autonomous University of Guadalajara, the principal source of Fifth Pathway applicants; (2) high enrollment of U.S. citizens in Caribbean medical schools whose students and graduates are not eligible for Fifth Pathway programs; and (3) low pass rates of U.S. foreign medical graduates on the Foreign Medical Graduates Examination in the Medical Sciences (FMGEMS) administered by the ECFMG. Successful completion of FMGEMS is required for entry into most Fifth Pathway programs.

The Council has requested officials at Guadalajara to send enrollment data for United States citizens; that request has not yet been fulfilled. The September 1985 Report of the U.S. General Accounting Office (GAO), *Federal, State, and Private Activities Pertaining to U.S. Graduates of Foreign Medical Schools,* estimates that 13,000 to 19,000 U.S. citizens are currently enrolled in foreign medical schools. A GAO report in 1980 on the same subject estimated U.S. citizen enrollment at 10,000 to 11,000. The majority of current enrollees are presumed to be attending medical schools in the Caribbean.

TABLE 1

ACTIVITY OF FIFTH PATHWAY PROGRAMS

Academic Year	Number of Applications Received	Number of Students Admitted	Number of Students Completing Programs
1973-74	197	126	122
1974-75	256	196	191
1975-76	764	274	262
1976-77	1,339	365	357
1977-78	2,400	476	399
1978-79	2,854	515	461
1979-80	2,459	547	516
1980-81	2,709	491	474
1981-82	2,444	510	506
1982-83	1,676	388	392
1983-84	1,912	391	403
1984-85	1,969	374	370
1985-86	1,345	208	199

The Council's interpretation of Fifth Pathway enrollment declines was confirmed in the informal conversations with program directors during the telephone survey. Some directors added that the overall quality of Fifth Pathway entrants is now higher.

Conclusions

Enrollment trends suggest that Fifth Pathway programs will continue to contract and that the number of sponsoring medical schools and programs will decline further. The Council on Medical Education discourages the development of new Fifth Pathway programs. Further, the Council believes that states with mandatory programs should review the advisability of these mandates as they consider health manpower issues.

Forty-one state licensing boards and the licensing board of Puerto Rico are willing to accept Fifth Pathway graduates as candidates for licensure, provided other qualifications are fulfilled. Recognizing the need to facilitate the interstate movement of licensed physicians, the Council believes that these licensing boards should continue to accept Fifth Pathway graduates as candidates for licensure.

In light of the Fifth Pathway enrollment trends described in this report, the Council on Medical Education does not perceive a need at this time to recommend any changes in AMA policy on the Fifth Pathway.

Management of Traumatic Chest Wall Defects

DAVID B. REATH, M.D.

Introduction

For nearly 100 years, reconstructive surgeons have wrestled with the problem of chest wall reconstruction.[1] Clarification of the vascular anatomy of various muscles and their overlying cutaneous territories has allowed several methods of managing acute and chronic chest wall defects. Although these methods are most usually applied to neoplastic or iatrogenic defects, they are equally applicable to acute chest wall trauma, as seen in the following case history.

Case Report

A 34-year-old man was transported by helicopter to the University of Tennessee Hospital at Knoxville after suffering a close range gunshot blast to the left anterolateral chest. In transport an occlusive dressing was applied and the patient received intravenous fluids and high flow oxygen. On arrival to the emergency department at 9:27 PM, the patient was hemodynamically stable. X-rays of the chest showed a small amount of shot within the chest and a pulmonary contusion (Fig. 1). The patient was quickly taken to the operating room where the lung was debrided and repaired (Fig. 2). Further debridement of the chest wall left a full thickness defect of rib, chest musculature, subcutaneous tissue and skin (Fig. 3). To close the defect a latissimus dorsi myocutaneous flap was

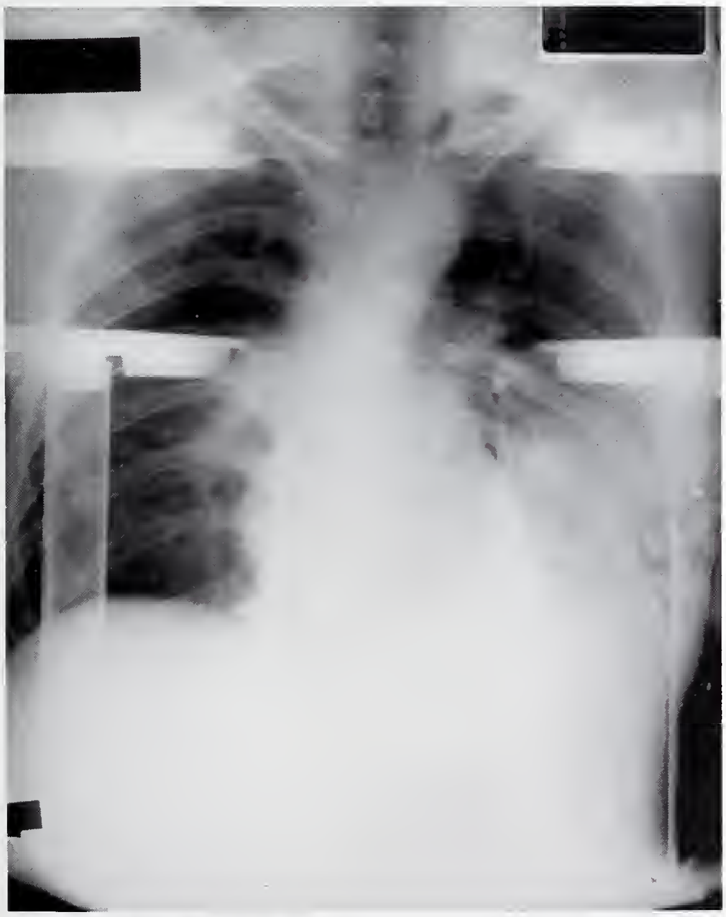

Figure 1. Admission chest film showing lung contusion.

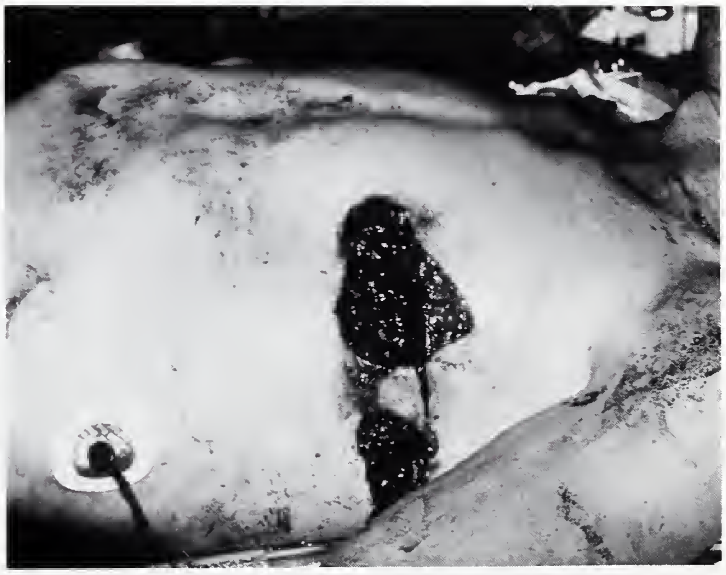

Figure 2. Appearance of chest wall injury prior to operation.

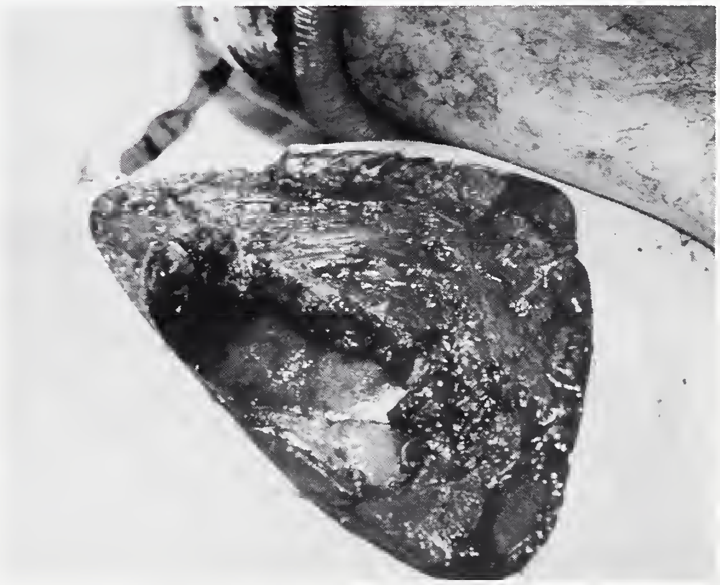

Figure 3. Chest wall defect following wide debridement.

From the Division of Plastic Surgery, Department of Surgery, University of Tennessee Hospital at Knoxville.

designed and elevated (Fig. 4). The flap closed the chest wall and restored its integrity (Fig. 5). The donor site was closed primarily. Postoperatively, the patient had an uneventful course and was discharged on the 14th day following operation (Fig. 6).

Discussion

The treatment of this patient's chest wall injury involves two principles. The first and most important of these is wide debridement of all contaminated and injured tissues. Hoping to minimize the soft tissue defect, surgeons are often not aggressive enough in debriding the wound. When one is dealing with a significant blast injury, tissue of marginal viability will become nonviable after 24 to 48 hours, and the preferred course is adequate debridement until viable tissue with good bleeding is observed in all areas of the wound.

The second principle of treatment involves reconstruction of the soft tissue defect. Whereas with injuries of the extremities, or even the abdominal wall, it is possible to delay reconstruction, particularly in the presence of significant wound contamination, with chest wall injuries, immediate reconstruction is preferred in order to restore the dynamics of normal respiration and ventilation.

In 1950, Campbell[2] first described the use of a latissimus dorsi muscle to close a full thickness wound of the anterior thorax. Unfortunately, this method of chest wall reconstruction went unappreciated for many years. In the past 15 years, the study and definition of muscle and myocutaneous flaps has made them some of the most reliable and useful reconstructive tools, particularly for problem wounds. Presently they are commonly used to reconstruct soft tissue defects in almost any anatomic location.

Though the major advances in chest wall reconstruction have dealt with defects due to neo-

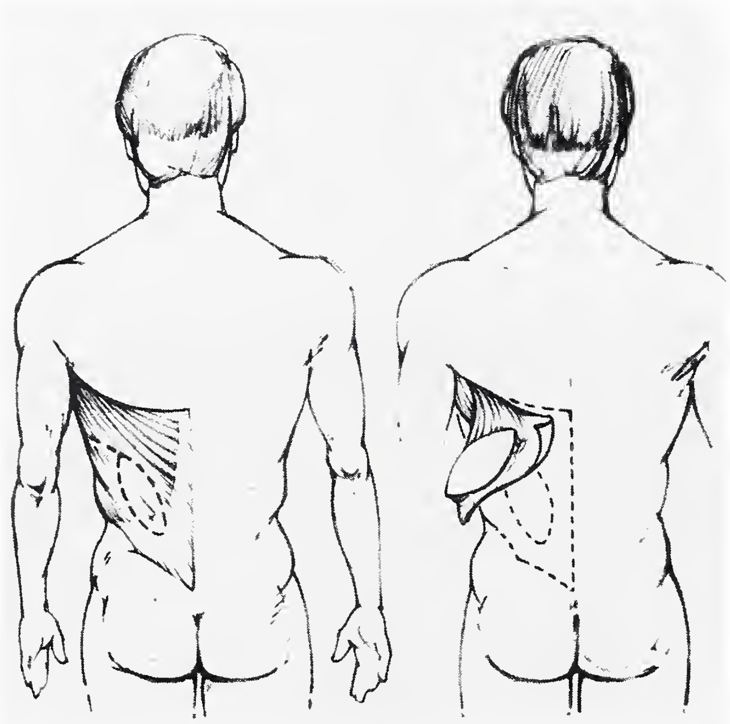

Figure 4. Latissimus dorsi myocutaneous flap design and elevation.

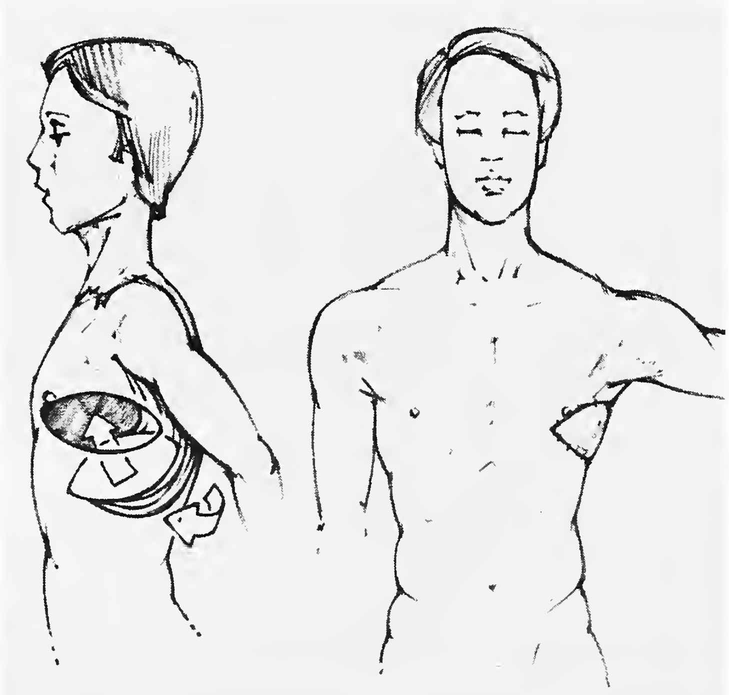

Figure 5. Completed chest wall reconstruction using flap.

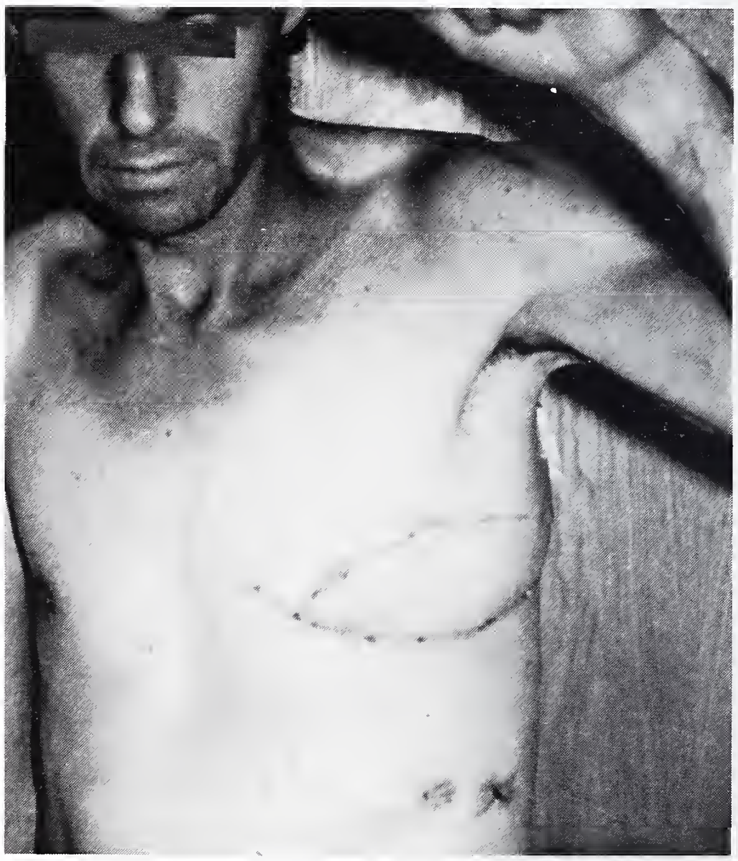

Figure 6. The patient at discharge 14 days following repair.

plasm, infection, or radiation, traumatic defects can be reconstructed by the same methods. Muscles with and without their overlying skin that are frequently used in chest wall reconstruction include the latissimus dorsi, the pectoralis major, and the rectus abdominis.

The reliability and versatility of the latissimus dorsi muscle and myocutaneous flaps were demonstrated by McCraw[3] and Bostwick[4] in the late 1970s. The muscle originates from the lower sixth thoracic vertebrae, the thoracodorsal fascia, and the posterior iliac crest, and has a tendinous insertion on the humerus. The dominant blood supply is from the thoracodorsal artery. In addition, there are several segmental perforators from the intercostal and lumbar arteries, which supply a medially based flap. When based on the dominant blood supply, the arc of rotation easily reaches the midline of the chest. Functionally, the muscle acts as an adductor, extensor, and medial rotator of the humerus and, as such, is expendable.

In addition to their reconstructive potential, muscle flaps provide better resistance to infection in contaminated and chronically infected wounds than other methods of wound closure. Although the reason for this is not known, it is presumed that the increased vascularity of the muscle and the ability to obliterate large areas of dead space are important.[5] In this patient, the chest wall was heavily contaminated. The uneventful healing of the wound was due not only to the adequate debridement, but to the use of a muscle flap and its ability to heal contaminated wounds.

Conclusion

Large traumatic defects of the chest wall may require immediate closure using a muscle or myocutaneous flap. A number of muscles, including the latissimus dorsi, are available for chest wall reconstruction. If aggressive debridement of a contaminated wound is followed by reconstruction using a large muscle flap with a healthy blood supply, rapid and uneventful healing will usually follow.

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SUPPORT PROJECT USA

Project USA is an AMA program to recruit fully licensed physicians for short-term, general medicine assignments at Indian Health Service (IHS) and National Health Service Corps (NHSC) hospitals and clinics.

The majority of vacancies are for two weeks, with some extending to four weeks. Participating physicians receive $750 a week at IHS facilities, and $500 a week at NHSC clinics. The doctors are reimbursed for round trip transportation expenses, and living accommodations are provided.

Interested physicians should contact John Naughton, Director, Project USA, American Medical Association, 535 N. Dearborn, Chicago, IL 60610; Phone (312) 645-4702.

VUH Morning Report

Airway Obstructions

Case Report

An 80-year-old white woman arrived at the VUH emergency room with a 12-hour history of severe sore throat of abrupt onset, followed in six hours by a "weak voice" and difficulty in swallowing. Four hours prior to admission she began to have difficulty breathing. Her oral temperature was more than 101.8° F at home, and she had shaking chills.

In the emergency room her respiratory rate was 26/min, and intermittent inspiratory stridor was audible. Axillary temperature was 98.8°F. She was able to swallow her own saliva with difficulty. Examination revealed a swollen uvula, bilateral tonsilar swelling, and anterior cervical adenopathy. The lungs were clear, and examination of the heart showed only a soft ejection murmur.

Laboratory examination showed a WBC count of 12,100/cu mm with a predominance of segmented neutrophils. Arterial blood gases on 4 liters of oxygen were pH 7.32, Pco$_2$ 51, and Pao$_2$ 101. Chest x-ray was unremarkable, and lateral neck film was remarkable for a moderately swollen epiglottis and an increased retropharyngeal space.

The patient was admitted to the intensive care unit, where after blood cultures were drawn she was treated with cefotaxime and humidified oxygen. An otolaryngology consultation was obtained.

Fiberoptic laryngoscopy showed swelling and erythema of the epiglottis and aryepiglottic folds. Because the airway was widely patent, a course of close observation was chosen. Thirty minutes after laryngoscopy, respiratory distress increased. Emergency intubation was complicated by hypotension and bradycardia. Subcutaneous emphysema and a pneumomediastinum occurred, but both resolved spontaneously. Betalactamase producing *Hemophilus influenzae* grew in all blood cultures. The patient experienced rapid improvement and was extubated on the third hospital day, recovering fully.

Discussion

The causative organism of adult epiglottitis is frequently not found, although *Hemophilus influenzae* has been isolated from a minority of patients. Staphylococci and streptococci have also been implicated as causative agents. Inability to consistently isolate bacteria has led to speculation about a viral cause of epiglottitis.

Severe sore throat, muffled voice, drooling, and stridor should all be clues leading to suspicion of epiglottitis,[1] the diagnosis of which can be confirmed by soft tissue lateral neck films or laryngoscopy. The findings in this patient of epiglottic and aryepiglottic swelling on laryngoscopy are characteristic.

Unlike the course of epiglottitis in children, the course in adults is frequently benign because of the larger size of the adult airway.[2] While much controversy continues regarding prophylactic intubation or tracheostomy in adults,[3] a reasonable course of therapy is careful observation in an intensive care unit for those patients who do not have severe respiratory compromise.

Therapy should include antibiotics directed against *H. influenzae*. Ampicillin has been the drug of choice, but the emergence of beta-lactamase producing strains now dictates use of chloramphenicol or a third generation cephalosporin. The use of corticosteroids is advocated by some, but is of unproven value.[4]

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Airway Obstruction

Case Report

An 89-year-old white woman in previously good health except for a nontoxic multinodular goiter developed acute severe shortness of breath while at rest; it was worse when she was supine. When she rapidly became mildly cyanotic and confused, an ambulance was summoned and she was transported to her local hospital where she underwent a difficult endotracheal intubation with #7 oral endotracheal tube. Intubation was complicated by an episode of ventricular tachycardia. She denied chest pain, fever, chills, cough, or hoarseness. Because of high airway resistance, ventilation was difficult even with a volume-cycled ventilator, and she was transferred to our care.

Examination on arrival revealed an alert elderly white

Prepared by Arthur P. Wheeler, M.D., Hugh J. Morgan chief medical resident, Vanderbilt Medical Center, Nashville.

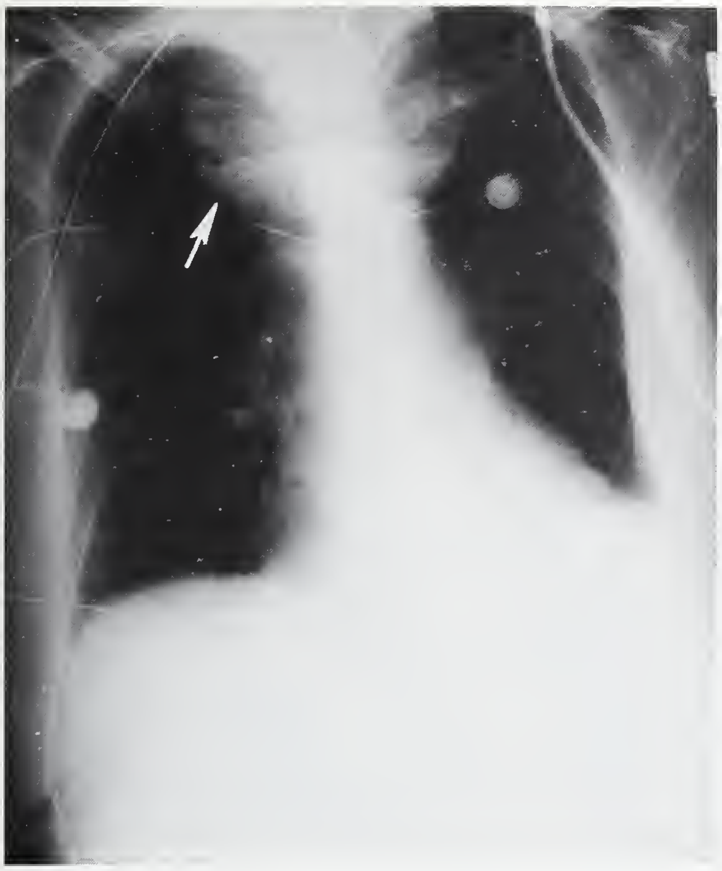

Figure 1. Smooth lobulated appearance of goiter extending to the anterior mediastinum.

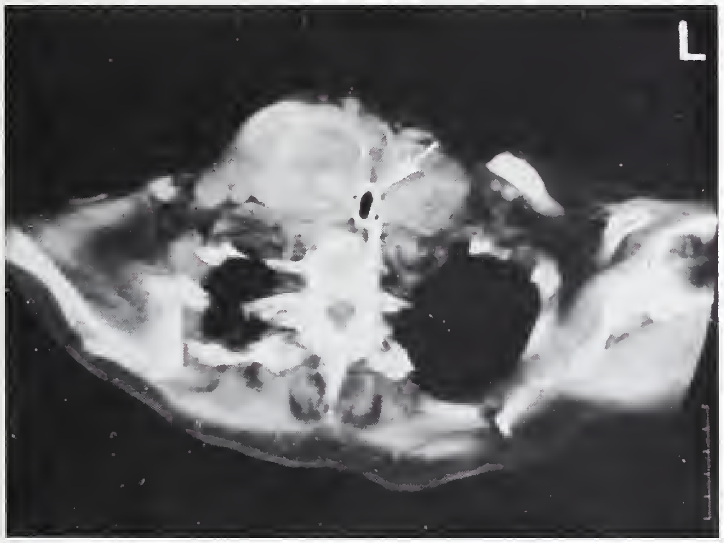

Figure 2. CT scan of the neck demonstrating heterogenous density of multinodular goiter and endotracheal tube compression by mass (arrow).

woman having normal vital signs and being ventilated mechanically. A large nodular goiter extending from the level of the hyoid bone to the suprasternal notch could be felt. Localized wheezing was heard over the lower third of the sternum. X-ray examination of the chest (Fig. 1) revealed a large smooth superior mediastinal mass. Electrocardiogram showed changes suggestive of an acute anterior myocardial infarction. Arterial blood gases showed normal oxygenation. Thyroid function tests were normal. A CT scan (Fig. 2) revealed a large multinodular goiter causing tracheal compression. It was also noted that the endotracheal tube was being deformed by extrinsic compression.

A needle aspiration biopsy of the mass revealed no evidence of malignancy. Enzymes failed to show evidence of myocardial infarction.

Because of progressive respiratory compromise, on the third hospital day surgery to remove the goiter was deemed necessary, even in light of the EKG changes, and complete removal of the goiter was accomplished without difficulty.

Discussion

Thyroid masses constitute some of the most common mediastinal tumors. Most mediastinal thyroids have a direct connection with the thyroid gland in the neck, and are nodular colloid goiters, whereas carcinoma is rare.[1] Seventy-five to eighty percent of intrathoracic goiters are from the lower pole of the thyroid, and extend into the anterior mediastinum, as in this patient. Goiters can also extend into the posterior mediastinum.[2,3] These masses are often smooth and well encapsulated, and show areas of cystic degeneration and calcification. By x-ray the goiters almost always appear to be smooth, lobulated masses of homogenous density displacing the trachea posteriorly and laterally. While most patients with goiters are asymptomatic, dyspnea can be produced by tracheal compression or by paralysis of the vocal cords due to compression of the recurrent laryngeal nerve. Obstruction of major mediastinal vessels can also occur.

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MRI Case of the Month

MICHAEL C. GAROVICH, M.D.; CHARLES L. ROBINETTE, JR., M.D.; and
TIMOTHY SCHOETTLE, M.D.

History

A 32-year-old white man was being examined for upper extremity weakness and wasting beginning four months earlier; prior to this he had been asymptomatic. A myelogram performed at another institution demonstrated an intramedullary mass lesion. MRI scan was then done to further evaluate the cord. Please evaluate Figs. 1 through 3 and see if you can arrive at the correct diagnosis.

Discussion

The advent of MRI (magnetic resonance imaging, formerly called NMR) has radically changed our approach to evaluating suspected cord lesions. Because of its inherent properties, MRI can directly image the cord without the bony artifacts involved in CT spine imaging. It also has the advantage over metrizamide studies in that it is not invasive, and directly rather than indirectly demonstrates the cord. Another significant advantage to MRI over other forms of imaging is that it allows examination in multiple planes, e.g., sagittal or coronal. The present case demonstrates a long cystic area within the cord extending from the upper cervical region down through the upper thoracic spine. What was thought to be a primary intramedullary neoplasm of the cord proved to be syringohydromyelia (syrinx). This study also demonstrates cerebellar tonsils lying below the foramen magnum (Chiari I malformation), which is frequently associated with syrinx.

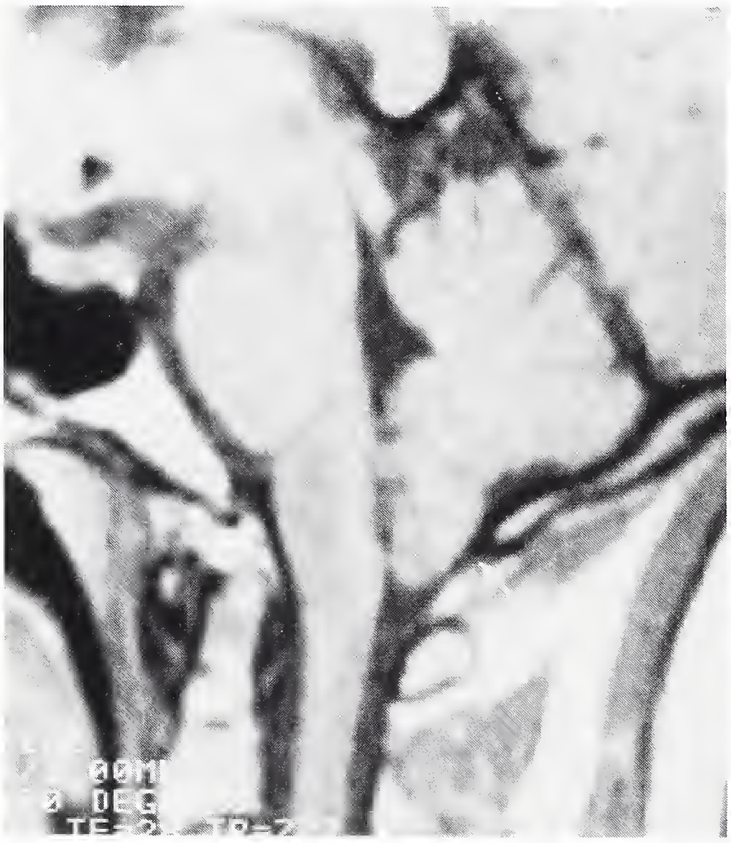

Figure 1. The cerebellar tonsils (arrow) extend below the foramen magnum. The brain stem and upper cervical cord are well demonstrated.

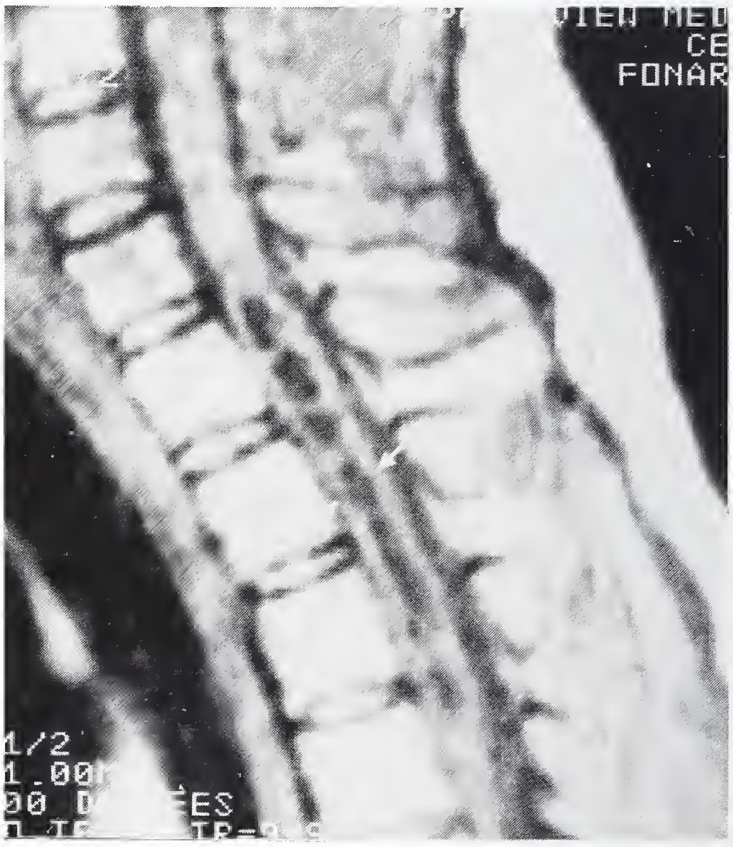

Figure 2. The cervicothoracic portion of the patient's syrinx appears as a region of low intensity within the cord on this T_1 weighted image (arrows).

From the Departments of Radiology (Drs. Garovich and Robinette) and Neurosurgery (Dr. Schoettle), HCA Park View Medical Center, Nashville, TN 37203.

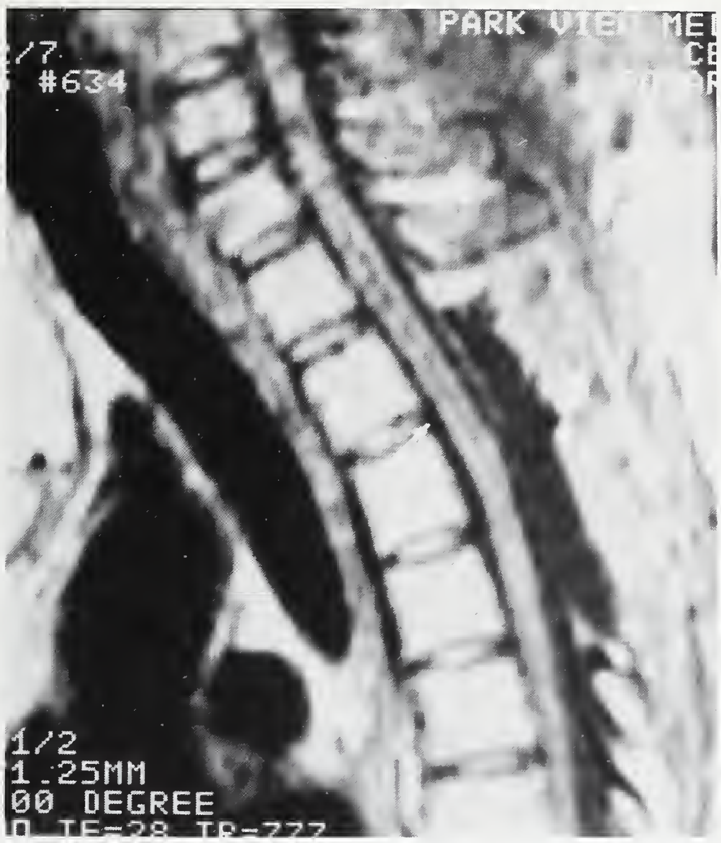

Figure 3. Following decompression surgery, the syrinx has collapsed (arrow).

Although such patients may be seen early in life, they frequently do not appear until adolescence or early adulthood. They often show cord symptoms, which are frequently confused with multiple sclerosis.

Myelography will demonstrate widening of the cord suggesting an intramedullary lesion. In the past, gas myelography was performed in an attempt to collapse the cord about the cystic structure. CT scans can demonstrate a syrinx following metrizamide, and delayed scans may show contrast medium in the cystic structure. CT, however, frequently has problems with bony artifact, and with CT patients cannot easily be studied directly in different planes. Finally, MRI has the advantage of requiring ionizing radiation. Although it varies from institution to institution, the cost of an MR scan at our institution is approximately the same as a CT scan.

ANSWER: Chiari I malformation with associated syrinx. The patient underwent surgery with decompression of the cord cavity, and progressed satisfactorily postoperatively.

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Health and Environment Report

Obstetrical Services in Tennessee

FREDIA S. WADLEY, M.D., MSHPA

Across the nation and within Tennessee, people are beginning to ask, "Who will deliver the babies?" This question is being asked because physicians and hospitals in rural communities are deleting obstetrical services.

It is estimated by Department of Health and Environment surveys that over the past four to five years, at least 20% of Tennessee physicians providing obstetrical care have either deleted this service entirely, decreased their obstetrical caseload significantly, or performed only emergency deliveries. Another 5% to 9% have indicated they are considering similar action. Although many reasons are given for this trend, most physicians agree that the rising insurance premiums and the number of malpractice cases related to obstetrical care are the straws that broke the camel's back.

Over the past two to three years, another facet of this problem has surfaced. Several hospitals in the northern part of the East Tennessee region have stopped providing deliveries and are now seeking approval to officially delete obstetrical services. Approval to delete obstetrical service must be obtained from the Health Facilities Commission through the Certificate of Need process, and the Board for Licensing Health Care Facilities must grant the hospital a waiver, since obstetrical services are mandated for general hospitals. A waiver may be granted by the Board if deletion of this service does not pose a threat to the health of the community.

Hospitals are giving two reasons for deleting obstetrical services. First of all, this service is sometimes a money loser in small rural hospitals. Maintaining staff, facilities, and equipment for a small volume of deliveries, when up to 40% of the patients may be indigent, often results in red ink. Secondly, hospitals are finding it more difficult to recruit physicians to provide obstetrical care. Obstetricians primarily locate in cities with a population of 30,000 to 40,000 or greater. In the more rural counties, family practitioners and general practitioners have provided most of the obstetrical care. Unfortunately, new recruits are less likely to include obstetrics in their practice due to time constraints and malpractice problems, and possibly a lack of emphasis during their training.

It is not surprising that some hospitals and physicians are less than eager to provide obstetrical services, but one has to wonder where this trend will lead us.

- Hospitals in neighboring counties might be put in the position of providing a greater number of deliveries to out-of-county indigents.
- With no physician providing obstetrical care, there is a greater risk that indigent women will not receive adequate and early prenatal care. This could lead not only to increased infant morbidity and mortality, but also to a greater need for very costly perinatal services.
- If the unavailability of obstetrical services increases, will entrepreneurs fill the gap with birthing centers, which can feed problem cases to a centrally located obstetrician?

In May 1986, the Department of Health and Environment increased the Medicaid reimbursement for obstetrical services (global $650 to $725) in an effort to ease the burden on those physicians with greater Medicaid caseloads. The Tennessee General Assembly will be considering this session an option to provide Medicaid coverage to pregnant women whose income is less than 100% of the poverty level. This could ease the

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From the Tennessee Department of Health and Environment, Nashville. Dr. Wadley is chief medical officer of TDHE.

financial burden of the physicians and hospitals by covering almost 6,000 additional pregnancies. Tennessee legislators will also deal with tort reform this session, but any benefits from these changes would be of a long-term nature and probably would have no great immediate impact on this problem.

Because there are too many factors involved for a simple solution, no one has an easy answer for the developing shortage of obstetrical manpower and facilities. The Board for Licensing Health Care Facilities has asked that a group be appointed to study the problem and keep them informed, since other waiver requests are expected. This study group will have physician and hospital representation, and it is hoped that they suggest means that will help alleviate the obstetrical shortage.

The most important concern right now may well be how to entreat physicians and hospitals who are presently providing obstetrical care to continue this important service. As one hospital administrator said at the Southern Legislative Conference last November in Nashville, "The decision to deliver babies is not made for financial reasons—but for medical reasons."

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Loss Prevention Case of the Month

Consider First Things First

J. KELLEY AVERY, M.D.

Case Report

A 67-year-old woman, who had been previously diagnosed as having an esophageal hiatal hernia, was seen in the emergency room complaining of chest pain which began following her evening meal two days before. She was admitted by the emergency room physician with orders for a barium swallow and surgical consultation.

The barium swallow, done early the next morning, demonstrated a fairly high-grade esophageal obstruction by a 2.8-cm foreign body located above Schatzki's ring in the distal esophagus. The patient was then taken to the operating room where, under general anesthesia, the esophagoscope was passed without undue difficulty and a moderate amount of meat was removed in multiple sections with the foreign body forceps. Afterwards, the esophagoscope was passed to its entire length, apparently into the hiatus hernia. Following the procedure, the patient was returned to her room in an alert and responsive state.

Approximately 30 minutes later the patient complained of severe chest pain, for which she was treated with nitroglycerin sublingually without relief. She was immediately seen by a cardiologist, who, following a cursory examination, reached the diagnosis of possible pleurisy, and the patient was transferred to the intensive care unit for overnight observation; a portable chest x-ray revealed possible pneumonia, but also demonstrated what appeared to be a foreign body in the distal esophagus.

On morning rounds the patient was found to be restless, dyspneic, and with diffuse chest pain. Repeat chest x-ray revealed subcutaneous emphysema in the neck and over the upper chest wall. There was also some left pleural effusion that appeared to be located posteriorly. Serial chest x-rays and arterial blood gases were done for the next three days, whereupon, with the patient being five days post-esophagoscopy, the possibility of perforation was entertained. After a chest tube was placed in the left posterior chest, evacuating approximately 200 cc of purulent fluid, the patient's chest x-rays improved, but her arterial blood gases remained marginal.

The sepsis associated with the perforated esophagus failed to respond to antibiotic therapy, and the patient ultimately progressed to irreversible cardiopulmonary arrest. Autopsy revealed acute mediastinitis and pleuritis with bilateral pleural effusions due to a traumatic laceration of the distal esophagus.

Loss Prevention Comments

In reviewing the essential features of the case, one is prone to react with dismay that the possibility of perforation of the esophagus did not occur to the attending physicians much earlier. The factors that seem to have contributed to this delay in diagnosis were several, which, again, in retrospect seem hard to explain.

First, the chest pain 30 minutes following the endoscopic examination was clinically typical of myocardial infarction, with sweating, pallor, transient hypotension, and classical pain radiation. To further complicate the picture, there was an EKG which showed changes which when compared to a previous tracing suggest this. Though the portable chest x-ray showed only "possible pneumonitis," a pulmonary embolus was seriously considered. Apparently no other possibilities were entertained.

Second, the chest x-ray done the second day showed subcutaneous emphysema, which was overlooked, presumably due to a delay in getting the radiologist's written report into the record. (The films were taken Friday morning; the written report was typed that day, but was not seen by the surgeon until Monday, the fifth day post-endoscopy.)

Third, and probably the primary reason for a large financial settlement, was that the surgeon had had little previous experience in esophagoscopy, and in deposition had to admit that he had never seen subcutaneous emphysema before. Recognizing the incomplete removal of the meat, he attributed the patient's symptoms to either the retained foreign bodies or some cardiopulmonary complication.

Finally, there was no documented evidence that there was any real communication between the physicians involved in the management of this unfortunate patient. Real coordination of medical effort in this case probably would have resulted in an earlier diagnosis and would possibly have prevented the tragic outcome. This apparent lack of coordination of physician efforts occurs with alarming frequency in medical practice. Don't let it happen to you!

Dr. Avery is medical director for Continuing Medical Education, St. Thomas Hospital, Nashville.

president's page

James R. Royal

Being a Physician
What Do You Do?

All of us at some time, on being introduced, have been asked "What do you do?" We usually respond with the generic "I'm a doctor" or "I'm a physician." But when meeting another physician and asked the same question, we get more specific and answer with our specialty or area of practice. We become family physicians, surgeons, pathologists and all the other specialties. We know what we mean when we answer the question "What do you do?"

When we ask ourselves "What do I do?" we may have difficulty answering. Being a doctor and practicing medicine is a complex and hard to precisely define profession. There are probably as many answers as there are doctors—no two being exactly alike.

The long years of preparation to become a doctor are more demanding than for any other profession. They are necessary but we must realize that the system exacts a toll. Early on in pre-med and medical school the students are separated from their peers in other fields of study. Personal contacts are progressively narrowed until in residency they are almost exclusively with those in the same fields of study. This prolonged childhood and limited association tend to narrow our viewpoints. Somewhere, sometime, and somehow we learn to focus on the problems of others—our patients.

To be a doctor is more than repairing deformities, correcting chemical imbalances, or curing infections. It is becoming involved with the patients' problems, their families, their jobs, their total environment.

We find ourselves involved in myriads of situations, all unique to the particular patient. Each situation requires its own solution. Coping with a defective newborn is quite different from caring for an elderly patient with terminal cancer. As we assume responsibility for patients' medical problems, we often become involved in their other problems—socioeconomic, marriage, drugs or alcohol, smoking, and others.

The practice of medicine is not just a way to make a living. To be referred to by a patient as "My doctor" is a special tribute and trust, and must be earned and cherished. When we ask ourselves "What do I do?" maybe saying "I'm a doctor" says it all.

James R Royal, MD

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FEBRUARY, 1987

editorials

TMAHMSS

One of the occasional though major frustrations of periodical publishing is that a periodical is just that—periodical, with all the limitations implied by the term. People have become so accustomed to receiving news even while it is still in the making that the notion of lead times often fails to filter through their reticular network, and we receive requests for our services, even from some who should know better, that we cannot possibly accede to. One would think, in this day of electronic wizardry, that a letter written December 8 would provide more than adequate time to let our readers know about an event on March 1, almost three months away. It doesn't.

Discounting occasional (or maybe more than occasional) vagaries of the postal service (on December 18 I received two important pieces of first class mail postmarked December 9 and December 11—sent from three blocks and two miles away, respectively), announcements, in order to be useful, should reach us four months before the event—clearly not possible in some instances, but also clearly possible in other instances when the correspondents are simply dilatory.

I'm not sure which was the situation in the instance that prompted this piece, but we were requested on about December 10 to announce the organizational meeting of the newly formed Hospital Medical Staff Section of the TMA to be held on March 1, 1987. If you read your *Journal* the day it plops itself on your desk, which even were I the most optimistic of editors I should doubt, you might have a couple of weeks to prepare yourself. The hooker is that applications have to be received at the TMA office by February 20—about the time your *Journal* will be arriving, with any luck. Hotel reservations needed to have been in, though, by January 28. No luck.

It is fortunate that there are other means of communicating with you, as notification through the *Journal,* including this piece, is clearly late, and useless as an information item. This is written, therefore, not so much to inform as to exhort. There are two exhortations: first, to participate in your hospital medical staff activities, and to be sure your delegate to the Hospital Medical Staff Section is the individual who can best represent you; second, if you have not already, to join the AMA, which conceived and nurtured the notion of the Hospital Medical Staff Section.

When the proposal for such a section was placed before the AMA House of Delegates a couple of years ago, the reaction was, "Oh, no! Not another delegate!" You see, the House has nearly doubled in size in the past ten years, and is fast becoming unwieldy, even nigh unmanageable. The meeting of the section, to which the staff of each JCAH-accredited hospital can send a delegate, has proved to be a successful forum where the many problems peculiar to the hospital staff are aired and from which a number of significant resolutions have been forwarded to the House for action. A number of insignificant ones have, too, of course; but no matter. That number is diminishing as the section becomes more mature. After a couple of years at it, they are getting over feeling their oats.

One might question to what end all this activity—can it accomplish anything? After all, is anyone listening? That is not an unnatural response, I hope; it is one I ask myself each time I set out to attend a meeting of the AMA House, or a TMA House meeting or a meeting of the Board of the Nashville Academy of Medicine—or sit me down to write an editorial, for that matter. The only practical answer I know to the question that goes, "When a tree falls in the forest, is there any noise if there is no ear to hear it?" is that there certainly is no noise if the tree does not fall. Who knows who is listening?

One of medicine's biggest problems is the propensity of doctors to sit in their quiet offices on that cushion of apathy and pontificate that all they want is to be left alone to practice medicine. Don't we all! But what has that attitude—the prevailing one, I would add—gotten us? It has gotten us Medicare, PSRO, HMO, TEFRA, HCFA, and all sorts of governmental incursions into a profession (or "trade," as we are now considered—another self-inflicted wound) that is correctly perceived as fragmented and incapable of concerted action or political pressure.

The basic unit of organized medicine is the county medical society; it elects and sends delegates to the AMA House. The basic unit of medical practice is for the most part, and for most of us has become, the hospital medical staff, with its own particular set of problems. It was recognition of this fact that prompted formation of the AMA's Hospital Medical Staff Section by the AMA House of Delegates, with a recommendation that it be duplicated at the state level; hence the adoption by the TMA House last April of the amendment to the Constitution that culminated in this present action. It is simply another channel for introducing concerns onto the floor of the TMA House.

It is a sad commentary on our fractiousness, but more people are members of medical staffs than of their county societies. Though those nonmembers have excluded themselves from active participation at higher levels, they can at least have a measure of influence—as much as they can engender—into the process through their delegate to the Hospital Medical Staff Section (who must be an AMA and TMA member). This is more influence than in my estimation they deserve, but if it serves as a unifying influence, let's have at it.

Though I am not particularly sanguine about the likelihood that the house of medicine will achieve unity by any means or on any topic, since doctors are just people, and as individuals are divided by as many issues as people generally, nevertheless, the Hospital Medical Staff Section is one more avenue, and one closer to home, to air the concerns of doctors. Support your newly formed Hospital Medical Staff Section. Even better (though I realize that like the preacher I'm threatening the wrong crowd with hellfire) join organized medicine at all levels and become a participant instead of simply a spectator. Sure, it can be frustrating, but so can anything else—even getting out the *Journal*.

J.B.T.

Pathways to Practice

One of the issues confronting American medicine right now tends to get lost in the rush, what with professional liability problems and incursions by various agencies into the practice of medicine. I am speaking of problems surrounding the foreign medical graduate (FMG), which may or may not be of interest to you, though it occupies an inordinate amount of the attention of the AMA House of Delegates.

Let me say first that I hesitate to use the word problem here, or even the term FMG, as neither is uniformly perceived by either the FMGs themselves or the medical profession at large. Not everyone recognizes that the term FMG encompasses not only foreign nationals, but also foreign educated United States citizens of both U.S. and foreign birth. When most of us speak of FMGs, though, we are usually thinking of the foreign born and educated doctor.

Not everyone concerned believes there is a problem, either. Many FMGs in all categories have been absorbed into the mainstream of American medicine without fanfare; this includes virtually all of the U.S. born nationals, but it also includes a considerable percentage of foreign nationals as well. A significant number of FMGs of foreign birth are officers in their local and state medical societies, and some are members of the House of Delegates, both in Tennessee and in other states, as well as of the AMA House of Delegates.

Others in both camps perceive problems of varying magnitude. Some in the medical establishment (meaning those who are graduates of LCME approved medical schools) perceive problems of individual competence in some instances (I need not point out that of course not all LCME accredited graduates are equally and superbly competent, either) and of competition (when things get tight, the first victims are always those who are "different"). For their part, many FMGs believe they have found no acceptance and have no voice in American medicine.

As a personal aside, I have found the FMG, viewed dispassionately and generically, little different from my other colleagues. All are variably capable and competent, and equally subject to the Peter Principle. As with their non-FMG colleagues I find some more acceptable than others, based on other characteristics than their origins or their "FMGness." As usual, the bell-shaped curve applies.

In times of a doctor glut such as we now have, and with severe shortages in other countries, it makes no sense to import doctors of foreign origin. At the same time, nationals of many countries are subject to severe oppression, and choose the United States, perceived as the land of the free and the home of the brave, the "golden door," so to speak, as their haven. Many of them have suffered much. On grounds of either humanitarianism or Christian charity they need to be afforded whatever succor and opportunity we can offer. Increase in their numbers among us is presently small.

I have found that there is great confusion about how an FMG—either U.S. or foreign—can enter the system; there is particular confusion over the so-called "Fifth Pathway," especially as to what, if this pathway is the fifth, the other four are. A report from the AMA's Council on Medical Education (CME Report C, I-86) is a clear exposition of the whole matter, since though it is entitled "The Fifth Pathway," it is in fact a complete review of the means of entrance of the FMG into the practice of medicine in the United States, and their utilization of it. I therefore include it in this issue of the *Journal* for your information and edification.

As doctors face growing competition for patients, additional colleagues of *any* sort represent a threat to one's livelihood, and acceptance of any of them, let alone the FMG (who though he perhaps should be considered no differently, is) in this situation can be a real strain on humanitarianism and/or Christian charity. As I write this it is going on Christmas-time; you will read it somewhere around St. Valentine's Day. I hope it did not provide too cold a blast onto the feelings of expansiveness and munificence those special times engendered, and so quench your flame. If so, just button up and bear up, and expect added stars in your crown.

J.B.T.

Birthing Blight

If there is any satisfaction at all to be found in the gloom of today's medical practice climate, it is to watch the fog (too infrequently) roll out from our morass and spill over onto the general public. Though it is small comfort indeed, it is some. I take no delight otherwise in Dr. Fredia Wadley's report from the Tennessee Department of Health and Environment, carried elsewhere in this issue of the *Journal,* documenting the local manifestation of a phenomenon already widespread in other parts of the country, notably Florida.

There is nothing edifying, of course, in observing the compounding of human misery, which is what Dr. Wadley's report is about, but drastic means are often required for a cure. It has long been the perception of doctors generally that only when the situation got bad enough would legislatures be moved to get off their status quos and remedy the defects in the tort system. Now those defects have resulted in restriction of medical services to a large segment of the population, the one to which the pregnant woman belongs. To coin a phrase, Hell hath no fury like a woman scorned. The legislature just might pay attention to *them*.

As the government, both federal and otherwise, has discovered through tireless investigation and tinkering with the system, there are all sorts of ways to harass doctors and regulate the way we practice medicine. I believe there is no way, though, that they can make a doctor deliver a baby if he doesn't want to. I suppose in the worst case scenario (a term I detest) they could banish him to the Denali Wilderness, but we have not yet reached that barbarous totalitarian situation in this country. By the same token, it would be hard for the authorities to revoke a hospital's license simply because the hospital did not deem it feasible to maintain an obstetrical service—though of course they might do that, too.

Dr. Wadley comments that the most important concern is how to entreat doctors and hospitals to continue providing obstetrical services. I'm sure that Dr. Wadley, like the rest of us docs, knows it's real easy; she is just being coy. Convincing the legislature is the meat of the matter. Failure of a legislator or so to find a doctor to deliver his first grandbaby might help.

The cry of "sue the bastard" rings too loud and clear in the obstetricians' ears; humanitarianism falters under the threat of losing house and home.

J.B.T.

in memoriam

Thomas Keith Beene, age 48. Died December 7, 1986. Graduate of University of Arkansas School of Medicine. Member of Knoxville Academy of Medicine.

Robert S. Cowles, Jr., age 61. Died December 4, 1986. Graduate of University of Tennessee College of Medicine. Member of Greene County Medical Society.

Earl Dorris, age 85. Died October 12, 1986. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

Walter S. E. Hardy, Jr., age 66. Died November 28, 1986. Graduate of Meharry Medical College. Member of Knoxville Academy of Medicine.

Embry A. McKee, age 48. Died November 23, 1986. Graduate of University of Tennessee College of Medicine. Member of Nashville Academy of Medicine.

Gerald I. Plitman, age 61. Died December 18, 1986. Graduate of Vanderbilt University School of Medicine. Member of Memphis-Shelby County Medical Society.

Henry C. Smith, age 85. Died October 26, 1986. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

George Walker Sommers, Jr., age 56. Died November 20, 1986. Graduate of University of Virginia School of Medicine. Member of Lakeway Medical Society.

TMA Members Receive AMA Physician's Recognition Award

Nineteen TMA members qualified for the AMA Physician's Recognition Award during November 1986.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

George F. Bale, M.D., Memphis
Larry C. Collins, M.D., Cleveland
George L. Eckles, M.D., Winchester
Joseph H. Fishbein, M.D., Nashville
James W. Giles, M.D., La Follette
Steve A. Hyman, M.D., Nashville
Sarma R. Kunda, M.D., Chattanooga
Joseph C. Lougheed, M.D., Memphis
Rogelio D. Martinez, M.D., Nashville
Douglas O. Olsen, M.D., Brentwood
Kelly Patterson, M.D., Memphis
Diane J. Pincus, M.D., Antioch
Bill S. Portis, M.D., Huntingdon
Jack B. Pruett, M.D., McMinnville
W. Scott West, M.D., Nashville
Lester F. Williams, M.D., Nashville
Clarence C. Woodcock, Jr., M.D., Nashville
Richard L. Wooten, M.D., Memphis
Robert R. Yarbrough, M.D., Memphis

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

COCKE COUNTY MEDICAL SOCIETY
Steven Gore, M.D., Parrottsville

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY
W. Rees Buttram, M.D., Chattanooga
Christopher S. Copeland, M.D., Chattanooga
Patricia A. Green, M.D., Chattanooga
A. Lee Solomon, M.D., Chattanooga
Dennis J. Stapleton, M.D., Chattanooga
Steven B. Stubblefield, M.D., Chattanooga
Carl D. Yates, M.D., Chattanooga

NORTHWEST TENNESSEE ACADEMY OF MEDICINE
Lee Harris, M.D., Dyersburg
Thomas E. Boston, M.D., Dyersburg

RUTHERFORD COUNTY/STONES RIVER ACADEMY OF MEDICINE
Marvin Lewis, M.D., Murfreesboro
Stanley Paul Mullen, M.D., Murfreesboro

SULLIVAN COUNTY MEDICAL SOCIETY
R. Alan Davis, M.D., Bristol

SUMNER COUNTY MEDICAL SOCIETY
Robert E. McDaniel, M.D., Gallatin

WILLIAMSON COUNTY MEDICAL SOCIETY
Mary McCormick, M.D., Franklin

personal news

Robert P. Christopher, M.D., professor and chief of the Division of Rehabilitation at the University of Tennessee at Memphis, has been named president of the American Academy for Cerebral Palsy and Developmental Medicine.

Charles D. Huffman, M.D., Hendersonville, has received the Tennessee Public Health Association's first Alex B. Shipley Award, recognizing his outstanding contributions to public health.

Peter Reardon, M.D., Greeneville, has been certified as a Diplomate of the American Board of Obstetrics and Gynecology.

Joe S. Smith, M.D., Knoxville, has been elected a Fellow of the American College of Cardiology.

Willian N. Smith, M.D., New Tazewell, has received the Tennessee Hospital Association's Meritorious Service Award, recognizing his many years of community and public service.

The following TMA members have been elected Fellows of the American College of Surgeons: *William D. Bowers, M.D.*, *Jack P. Byrd, M.D.*, *John Stephen McNulty, M.D.*, all of Cleveland; and *Richard L. Schultz, M.D.*, Oak Ridge.

announcements

CALENDAR OF MEETINGS

NATIONAL

March 7-13	International Academy of Pathology, United States-Canadian Division—Palmer House, Chicago
March 8-12	American College of Cardiology—New Orleans
March 9-12	American College of Emergency Physicians—Loews Ventana Canyon Resort, Tucson, Ariz.
March 11-14	American Cleft Palate Association—San Antonio, Tex.
March 14-18	Congress of the International Anesthesia Research Society—Buena Vista Palace, Lake Buena Vista, Fla.
March 15-19	American Association for Pediatric Ophthalmology and Strabismus—Scottsdale, Ariz.
March 16-18	Southeastern Surgical Congress—Atlanta
March 19-22	Society for Adolescent Medicine—Seattle
March 22-25	Society of Air Force Physicians/American College of Physicians—Fairmont Hotel, New Orleans
March 25-28	American Society for Clinical Pharmacology and Therapeutics—Marriott's Orlando World Center, Orlando, Fla.
March 25-29	American Orthopsychiatric Association—Washington Hilton, Washington, D.C.
March 26-29	American Holistic Medical Association—Marriott Hotel/Sea-Tac, Seattle
March 26-29	American Psychosomatic Society—Sheraton Society Hill, Philadelphia
March 28-April 2	American Society of Clinical Pathologists—San Francisco Hilton
March 28-April 2	College of American Pathologists—San Francisco Hilton
March 29-April 3	American Association of Immunologists—Washington, D.C.
March 29-April 3	American Association of Pathologists—Washington, D.C.
April 2-7	American College of Physicians—New Orleans
April 3-5	International College of Surgeons, United States Section—Sheraton Washington, Washington, D.C.
April 4-8	American Society of Abdominal Surgeons—National Study Center for CME, Tampa, Fla.
April 6-8	American Association for Thoracic Surgery—Chicago
April 11-13	American Society for Laser Medicine and Surgery—Hyatt Regency Embarcadero, San Francisco
April 19-24	American Fracture Association—Chicago
April 23-27	American Medical Society on Alcoholism and Other Drug Dependencies—Stouffer's, Cleveland
April 25-26	American Laryngological Association—Fairmont, Denver
April 25-May 1	American Occupational Medical Association—Wyndham Franklin Plaza, Philadelphia
April 27-28	American Broncho-Esophagological Association—Denver
April 27-30	American College of Obstetricians and Gynecologists—Las Vegas
April 27-30	Southwestern Surgical Congress—Coronado, Calif.
April 27-May 1	Ambulatory Pediatric Association—Anaheim, Calif.
April 27-May 1	Society for Pediatric Research—Anaheim, Calif.
April 28-30	American Laryngological, Rhinological, and Otological Society—Denver
April 28-30	American Society for Head and Neck Surgery—Fairmont, Denver
April 29-May 2	American Burn Association—Washington, D.C.
April 30-May 3	American Association for the History of Medicine—Philadelphia
April 30-May 3	Christian Medical Society—International Hotel, New Orleans

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The Lupus Anticoagulant

RONALD H. LANDS, M.D.

Introduction

The lupus anticoagulant (LA) is an antibody, usually of the IgG class, believed to be directed against the phospholipid component of the prothrombinase complex. It is so named because of its historical association with systemic lupus erythematosus (SLE), but it is now known to be associated with a variety of other clinical disorders. I will present a case seen recently at the University of Tennessee Center for Health Sciences, and review the incidence, diagnosis, and pathophysiology of this disorder.

Case Report

A 33-year-old white male truck driver complained of pain in his posterior calf that had first occurred six to eight weeks earlier. The pain was associated with progressive swelling over the next few weeks. He came to the Veteran's Administration Medical Center when the pain and swelling extended into his thigh. He had noted coincidentally over this same period at least one episode of pleuritic chest pain that was associated with a sensation of breathlessness.

He was treated for a presumed true-positive VDRL six months prior to this admission, and had been admitted to the surgery service in 1985 for left upper quadrant pain that was ultimately diagnosed as costochondritis; this resolved spontaneously. His FANA and VDRL were both found to be positive on that admission, and his coagulation studies were normal at that time.

Physical examination on admission revealed a healthy appearing man with a palpable spleen tip, and a right thigh that was 3 cm larger in circumference than the left. There were no joint deformities, effusions, or skin rashes.

The admission complete blood cell count was normal except for a platelet count of 82,000/cu mm. The chest x-ray showed an elevated left hemidiaphragm and blunting of the left costophrenic angle. A venogram of the right leg showed occlusion of the right superficial femoral vein.

Coagulation studies were ordered in preparation for anticoagulation. The patient's prothrombin time (PT) prior to anticoagulation was 14.3 seconds (normal 11.0 to 13.5). The partial thromboplastin time (PTT) was prolonged at 149.7 seconds (normal 30 to 93.6). A mixture of patient and control plasma revealed a persistent abnormality with a PT of 14.5 seconds and a PTT of 116.6 seconds. The thrombin time (TT) was 14.1 seconds (normal 14.3 to 17.1). Thromboplastin dilution assay was compatible with LA.

He was diagnosed at this point as having a deep venous thrombosis due to LA.

Discussion

Although it shares part of its name with systemic lupus erythematosus, this is not a prerequisite for acquiring lupus anticoagulant. Printed estimates of the incidence of LA vary, but it is generally accepted as the most common circulating anticoagulant diagnosed. It may be present in as many as 5% to 10% of all patients diagnosed as having SLE. In one large restrospective review of a 20-year collection of patients with circulating anticoagulants, 70% were of the lupus variety. Of this group, half had SLE and the other half had some other disorder.[1] The LA has been associated with a variety of drugs, such as chlorpromazine and procainamide.[2] It has occurred in a multitude of chronic inflammatory diseases and malignancies.[1] It has most recently been associated with the occurrence of *Pneumocystis* pneumonia in patients with AIDS, and it has been documented to reverse with the suc-

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From the Department of Medicine, Hematology Section, University of Tennessee College of Medicine, Memphis.

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cessful treatment of this infection.[3] It is, therefore, an acquired disorder, and in many cases, it is reversible.

The discovery of LA is usually a consequence of abnormalities noted in the activated partial thromboplastin time (APTT), since this test is the most likely to be abnormal. The PT is either normal or slightly prolonged, and the TT is normal. By mixing the patient plasma with an equal amount of normal plasma and documenting the persistence of the coagulation abnormality, the presence of an inhibitor can be established. Acquired inhibitors of coagulation that must be differentiated from LA include inactivators of factor VIII, factor IX, factor V, and inhibitors of fibrinogen and fibrin stabilization.

To separate LA from the other possible causes of an acquired circulating anticoagulant, the dilute thromboplastin test is used. This test exploits the observation that exponential decreases in the concentration of thromboplastin added to the patient's platelet poor plasma causes relatively longer clotting times than the same concentration of thromboplastin in normal control plasma. The diagnosis of LA is one that can be made with relative certainty.

The importance of this diagnosis lies not in its relationship to the logical consequences of anticoagulation but to the paradoxic thrombotic risks that are associated with it. In a series made up of patients with SLE without LA compared to a group of patients with both SLE and LA, the relative risk of a patient with LA developing a venous thrombosis was 13.7% per year. The relative risk for the development of an arterial thrombosis was 6.7%. The risk in patients with SLE alone was 1.7% and 1.2% respectively.[4]

Most of the thrombotic events involve the deep venous system of the legs. Arterial events usually involve the cerebral vasculature.[4,5] Small vessels are also involved, as evidenced in patients with SLE and LA who have been documented to have more extensive microthrombi in their renal biopsies than patients with SLE without the anticoagulant.[4]

No series has found bleeding to be a significant complication if LA is the only coagulation abnormality. If bleeding has occurred, it has always been associated with severe thrombocytopenia, a qualitative platelet dysfunction, hypoprothrombinemia or some other additional hemostatic dysfunction.[2] Major abdominal and cardiac surgery has been performed in these patients without complications,[6] and renal biopsy has been performed without excess bleeding.[4]

Any understanding of LA and its relationship to thrombosis will have to explain the generally lower platelet counts and the increased frequency of false-positive VDRLs that have come to be associated with this syndrome; 70% to 80% of all patients with LA will have platelet counts under 120,000/cu mm at the time of diagnosis.[4,7,8] In a direct comparison of patients with SLE without LA, 65% had platelet counts of more than 200,000/cu mm at the time of diagnosis.[4] The incidence of the false-positive VDRL is about 45% in patients with LA,[8] as compared to a reported incidence of only 12% to 20% in patients with SLE without LA.

Recently, the development of a radio-immunoassay (RIA) for the anticardiolipin antibody has contributed new insight into the pathophysiology of this disorder. This antibody is directed against a phospholipid found in cardiac muscle, which is the same phospholipid that reacts with the Wassermann antibody which is the functional basis of the VDRL test for syphilis. The presence of this antibody has now been documented to have a statistical relationship to the presence of thrombocytopenia in patients with SLE, as well as patients with immune thrombocytopenia.[9] It probably also explains the inordinate frequency of false-positive VDRLs that occur in this syndrome.

The exact role of the antiphospholipid antibody in the mechanism of thrombocytopenia and thrombosis remains elusive. Cross reactivity of one antibody with one or more phospholipids has been observed.[10] Antibodies specific to phospholipids known to be located in high concentration on the platelet membrane cannot be demonstrated to bind there in vitro.[10]

An antibody theoretically may interfere with phospholipid function at sites other than the platelet membrane. Antibodies that inhibit the synthesis of prostacyclin have been demonstrated to possess the anticoagulant activity in a patient with LA and recurrent abortions.[11] The implication is that the antibody may interfere with the endothelial production of prostacyclin by hindering the construction of arachadonic acid from tissue derived phospholipids.

A discussion of the treatment for this syndrome is always overshadowed by the urgency contributed by the presence of a clot. Anticoagulation with heparin for the short term is the generally accepted treatment for the patient with an active clot. The appropriate dose of heparin is an empiric decision since the APTT is abnormal by definition prior to treatment. The only review to address the issue found that at a dose of 1,000 units per hour by continuous infusion, there were no bleeding complications and no new thrombi formed.[7]

Treatment of the patient recovering from thrombosis should involve removal of any agent or treatment of any underlying disorder that is associated with the formation of LA. Consideration should be given to oral anticoagulation in the patient with this anticoagulant and recurrent thrombosis, but there are no studies definitively addressing this issue.

The treatment of patients with spontaneously occurring LA without a history of inappropriate clotting is much more problematic. The use of immunosuppressive drugs in some series does not alter the clotting studies or prevent the formation of clots.[8] In other series, the laboratory evidence of the anticoagulant disappears, and with it the propensity for clotting.[4] The only situation in which immunosuppression seems to be clearly beneficial is in the pregnant woman with LA; there is evidence that prednisone and aspirin may decrease the incidence of fetal wastage in this subset of patients.[12]

In summary, this is a syndrome that is defined by the presence of abnormalities in the phospholipid dependent coagulation tests such as the APTT. In spite of the prolonged coagulation times, it is not associated with abnormal bleeding, and in fact, it is associated with a paradoxic risk of thrombosis. Its pathophysiology is intricately related to the interaction of antibodies and phospholipids at a site, and in a way that has yet to be determined. It is to be hoped that information derived from the investigation of this interaction will delineate a more sophisticated method of treatment.

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RULING AFFECTS ADJUDICATION OF DISABILITY CLAIMS

The settlement of a class-action lawsuit against the Social Security Administation and the Tennessee Disability Determination Section (the agency responsible for processing Social Security Disability Claims for Tennessee residents) has resulted in significant changes in the adjudication of disability claims. The August 20, 1986 ruling of the U.S. District Court for the Western District of Tennessee states that greater weight should be given to the medical evidence provided by the treating physician. This means that the evidence from the treating physician will have greater impact in rendering the disability decision.

There are two changes required by this decision that may significantly affect the role of the treating physicians in the adjudication of disability claims. First, the Disability Determination Section will include a Medical Assessment Form with all requests for medical information. This form, to be completed by the treating physician, provides the source an opportunity to express how the impairment(s) restricts the patient's work-related activities. Second, if it is determined that a special examination or ancillary studies are needed, the treating physician will be given the opportunity to perform the examination or studies. These changes are expected to result in more equitable decisions.

The DDS Professional Relations staff is available to speak at medical society meetings to provide further information about the effects of this ruling. If interested in more details, please call 1-800-342-1117, ext. 6531 or if calling in the local Nashville area, 741-6531 and ask for Ed Anderson.

Treatment of a Complex Hepatic Injury

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Introduction

Treatment of complex hepatic injuries requires appropriate preoperative support and evaluation, utilization of challenging surgical maneuvers, and consideration and correction of severe complicating physiological derangement. When there is significant intrahepatic vascular injury and hemorrhage, the mortality exceeds 50%.

Case Report

A 20-year-old white male motorcycle rider who collided with a truck was immediately transported to the emergency room, hypotensive and in respiratory distress, where nasal O_2, intravenous (IV) fluids, MAST trousers, and a right thoracotomy tube were provided, and a Foley catheter inserted. Rapid infusion of crystalloid restored blood pressure. Intravenous cefoxitin sodium (Mefoxin) was given.

General survey revealed minor cutusions and abrasions, a laceration of the right leg, contusion of the right posterolateral chest, rib fractures and a treated pneumothorax on the right, and a distended, tender abdomen.

CT scan with enhancement demonstrated liver contusion and laceration with a large collection of blood about the liver, normal bilateral renal function, possible subcapsular hematoma of the spleen, and no evidence of major vascular injury.

At laparotomy, the only injury found was a liver laceration involving the right anatomical lobe and coursing obliquely through the anteroinferior and posterosuperior segments. The vena cava was not injured. Multiple blood vessels and bile ducts visible on the surface were occluded with metal clips. Bimanual pressure, and the Pringle maneuver (<20 min/episode) were utilized with continued efforts to control surface hemorrhage, but there were recurrent episodes of hypotension despite IV infusion of crystalloid, whole blood, fresh frozen plasma, and platelet packs. The gallbladder was removed, and the anterosuperior portion of the right lobe removed by finger fracture to reduce the bleeding surface. A metal clip was placed to occlude the right hepatic artery.

There was failure of clotting ("dilutional coagulopathy") at this stage, and the decision was made to pack the liver surface with laparotomy packs (six), and close the abdomen under pressure. The patient had received 22 units of whole blood, and an IV total of 25,000 cc. Urine output had been continuous and adequate.

The patient stabilized in the intensive care unit on a respirator with C-Pap and PEEP; serial H&H determinations; fluid resuscitation as determined by monitoring of CVP, and pulmonary wedge pressures; restoration of platelet counts, PT, and PTT with platelet packs and fresh frozen plasma; and appropriate antibiotic coverage.

Reexploration was accomplished uneventfully 16 hours after completion of the first procedure, with removal of the packs, debridement of the liver, positioning of a viable omental pack, and drainage of the liver area through the posterior gutter on the right.

The patient's postoperative course was amazingly uneventful, and he was discharged from the hospital after 23 days.

Discussion

The basis of successful treatment of trauma victims is early attention to respiratory and circulatory status. All measures should be initiated to achieve rapid stabilization so that early evaluation of injuries can be accomplished. CT scan gives the best information on intra-abdominal injuries resulting from blunt abdominal trauma.

At laparotomy, simple liver injuries are treated with suture and drainage.[1] More complex liver injuries, however, may require bimanual pressure,[1,2] control of hemorrhage or bile leak,[1,2] Pringle maneuver,[3] hepatic artery occlusion,[4] hepatectomy or hepatotomy,[2,5] and/or viable omental packs.[6] Appropriate antibiotic usage and adequate drainage is required.

Coagulopathy associated with massive IV therapy for treatment of intra-abdominal hemorrhage may require insertion of intra-abdominal laparotomy packs, and closure of the abdomen under pressure.[1,5,7] Correction of the coagulopathy with blood, platelet packs and fresh frozen plasma must be effected, with subsequent reexploration (in 12 to 24 hours) for removal of the packs.[8]

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Hematuria, Flank Pain, Allergy to Intravenous Pyelogram Dye and Use of Coumadin in Munchausen Syndrome

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A patient's presenting complaints and history naturally influence the differential diagnosis and subsequent hospital course chosen by the physician. What appears to represent a discrete symptom complex may actually reflect a cleverly planned deception. Such is the case in an individual with chronic factitious disorder with physical symptoms also known as Munchausen syndrome.[1] We describe such a patient with the triad of hematuria, flank pain, and allergy to intravenous pyelogram (IVP) dye.

Case Report

A 25-year-old female unemployed graduate nurse was referred by her endocrinologist in a distant state for evaluation of secondary amenorrhea for six years. Independent evaluations by an endocrinologist and by a gynecologist were unrevealing. The patient denied menstrual bleeding after administration of medroxyprogesterone alone or with a month of estrogen priming. Such findings were unusual, since a hysterosalpingogram showed a normal uterine cavity, and uterine biopsy revealed proliferative endometrium.

Her past medical history was notable for multiple admissions to hospitals in at least two different states for fever of undetermined etiology. Cultures were negative on many occasions, except for one instance of a *Streptococcus faecalis* bladder infection. She stated she often grew weary of the extensive negative evaluations and would leave the hospital refusing further studies. Perhaps most significant was her past history of recurrent hematuria and flank pain. Evaluation of this problem was always unrevealing, except for one occasion when she reportedly passed a calcium oxalate renal stone. However, diagnostic studies were always limited by an alleged allergy to IVP dye, which she stated caused her throat to swell. Eventually, the recurrence of her symptoms resulted in the tentative diagnosis of loin pain-hematuria syndrome[2] for which she was treated with coumadin.

On her current admission, she had hematuria and moderate left flank pain. Prothrombin time was prolonged to three times normal. Complete blood cell count and platelet count were normal. Initial evaluation focused on the reason for referral (i.e., amenorrhea). Prolactin was minimally elevated at 21 ng/ml (normal, <15 ng/ml), but it was normal at 12 ng/ml when the phenothiazine-antiemetic, which she had been taking at home for nausea associated with her flank pain, was withheld for 24 hours. Estradiol was high at 218 pg/ml, a level seen in the late follicular phase. She was given 100 μg of gonadotropin-releasing hormone (GnRH) intravenously. Baseline FSH was 14 mIU/ml, normal for the follicular phase (3 to 15 mIU/ml), but LH was elevated at 27 mIU/ml (normal 8 to 25 mIU/ml). FSH rose normally to 20, 23 and 21 mIU/ml at 20, 40 and 60 minutes after GnRH injection, respectively. However, LH rose excessively to more than 50 mIU/ml at every time point. These results are suggestive of impending ovulation, but also are consistent with polycystic ovarian syndrome. The only reasons for her not to have menstrual withdrawal bleeding after medroxyprogesterone are that she did not take the medicine or that she denied the bleeding.

As her endocrinologic evaluation neared completion and discharge was anticipated, she complained of increased left flank pain and hematuria. She stated that her pain was severe, and it required frequent administration of parenteral meperidine. A KUB x-ray showed no calcified calculi, and ultrasound examination showed no obstruction to the ureteral collecting system. The patient refused an IVP due to her alleged allergy to the dye. However, cystoscopy with retrograde pyelogram was performed and showed a recent anterior urethral excoriation and several lesions in the bladder wall mucosa which appeared to represent penetration type injuries. The retrograde study was normal. A long knitting needle was observed in her room. Since self-inflicted trauma was strongly suspected, she was confronted with this idea and denied such activity. Psychiatric consultation was obviously indicated[3] and suggested to the patient, but she refused to participate in such an evaluation. Narcotics were rapidly tapered, her flank pain and hematuria resolved, and she was discharged two days later.

Comments

An essential criterion for DSM III for the diagnosis of chronic factitious disorder with physical symptoms (Munchausen syndrome) is symptoms that are plausible and under enough voluntary control that multiple hospitalizations occur.[1] It seems quite likely that the patient's hematuria was factitious due to self-inflicted injury, that her flank pain was fictitious, that her amenorrhea and lack of estrogen/progestin induced withdrawal bleeding were due to noncompliance or misreporting, and that some, if not all, of the

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episodes of fever of undetermined origin were fictitious. In many aspects this case represents a classical example of Munchausen syndrome with respect to fluctuating symptoms, onset in early adult life, the patient's medical education, multiple evaluations by a variety of physicians, and dramatic presentation. Especially interesting is the fact that the coumadin prescribed for loin pain-hematuria syndrome paradoxically provided the patient an avenue for exacerbation of her hematuria.

Multiple etiologies of hematuria in individuals with chronic factitious disorder with physical symptoms have been reported.[2,4-6] Blood has been introduced into the urine by pricking the finger, biting the cheek, and spitting into the specimen or by lacerating the vagina. Suspicion of factitious illness is further heightened when the patient presents with the complex of hematuria, flank pain, and alleged allergy to IVP dye.[6]

Identification of such patients is critical to the initiation of appropriate psychiatric treatment. Unfortunately, the prognosis is often poor,[1] but the risk from unnecessary procedures, as well as the incapacitating lifestyle associated with frequent hospitalizations, justifies the use of psychiatric intervention. Furthermore, in this era of rising hospital costs and financial restrictions, evaluation of such patients can be incredibly time-consuming and expensive when they are not rapidly identified. Because detection is often difficult, communication with medical authorities involved in past hospitalizations can be extremely beneficial.

Acknowledgment

We wish to thank Ms. Shelia Gad for her secretarial assistance.

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APRIL 1987

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
			1	2	3	4
5	6	7	8 TMA 152ND ANNUAL MEETING Hyatt Regency Hotel—Knoxville	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30		

Fallopian Tube Prolapse Following Abdominal Hysterectomy

ANH H. DAO, M.D. and PETER S. CARTWRIGHT, M.D.

Introduction

Prolapse of the fallopian tube through the vaginal vault following hysterectomy has been sporadically reported in the literature, with only about 70 cases recorded up to 1985.[1,2]

When one considers that up to 650,000 hysterectomies are performed each year in the United States alone, this is indeed a very rare complication. Nevertheless, clinicians and pathologists must be aware of this condition in order to avoid erroneous diagnosis and sometimes unnecessary procedures. Most cases of tubal prolapse have followed vaginal hysterectomy, only 15 having been reported following an abdominal hysterectomy.[2-4] We report an additional case and a review of the literature of this topic.

Case Report

A 25-year-old woman underwent total abdominal hysterectomy with left salpingo-oophorectomy for chronic pelvic pain and endometriosis. In the immediate postoperative period she developed vaginal cuff cellulitis which was successfully treated with antibiotics. Approximately two months after the operation a watery vaginal discharge began, accompanied by dyspareunia and mild suprapubic pain. The discharge did not improve with douches, and speculum examination revealed at the vaginal cuff a 1.5-cm bud of friable red tissue, which resembled granulation tissue and was biopsied. The histology showed fronds of fallopian tube epithelium impregnated with acute and chronic inflammatory infiltrates (Fig. 1). Three months after her original procedure, the patient underwent a laparotomy with repair of the vaginal defect and reinforcement of the vaginal cuff. The right fallopian tube was excised. Her second postoperative course was uncomplicated.

Discussion

The prerequisite for the diagnosis of prolapse of a fallopian tube through the vaginal vault is the presence of an opening joining the peritoneal and vaginal cavities. This opening could be the result of faulty surgical technique or infection in the pelvis.[5] Failure to close the peritoneum and vaginal vault properly, and the use of gauze packs or drains in the cul-de-sac help create a hiatus through which the tube prolapses. Infection can weaken and separate suture lines and predisposes to tubal prolapse, with or without the formation of hematoma. In fact, it is the most important predisposing factor; of the 16 cases of tubal prolapse following abdominal hysterectomy, 10 cases were noted to have postoperative infection.

The characteristic symptom of tubal prolapse is persistent vaginal discharge, with or without

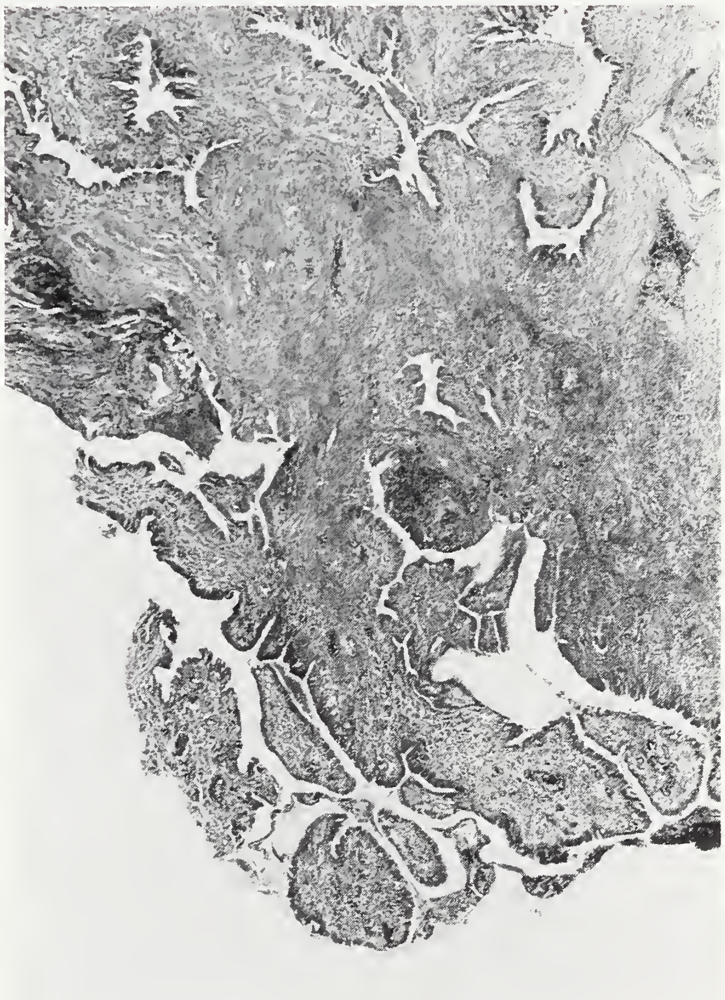

Figure 1. Fronds of tubal epithelium heavily infiltrated by inflammatory cells (hematoxylin-eosin, ×100).

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abdominal discomfort.[6] The discharge may be serous and odorless, or bloody and malodorous. Dyspareunia may be reported, as in our case. Uncommon symptoms include defecation with coitus, and pneumoperitoneum following intercourse.[2] The diagnosis of tubal prolapse is suggested by the symptoms and the presence of a bud of red tissue at the vaginal vault resembling granulation tissue, though usually not as friable. Passage of a probe into the tubal lumen is diagnostic, though not always possible.[7] The differential diagnosis includes papilloma, granulation tissue, endometriosis, prolapsed omentum or bowel, and malignant lesions. Usually, a biopsy is required.

Histologic examination of the biopsy shows tubal epithelium, usually infiltrated by acute and chronic inflammatory cells. The epithelial cells are often distorted and degenerate. Atypia may be present, and can be sufficiently striking[1] to confuse even the most experienced observer. Wheelock et al[2] recently reported a case thought to be an adenocarcinoma of the vagina in a postmenopausal woman.

That fallopian tube prolapse is more common following vaginal hysterectomy may be the result of leaving the vaginal cuff open following a vaginal hysterectomy but closed when the operation is performed abdominally. If opening the cuff is required to drain a hematoma or abscess, this might explain why these conditions are predisposing factors for the development of this complication. In our case, the vaginal cuff was not closed at the first operation, but rather, cuff hemostasis was obtained by using a circumferential locking stitch of polyglycolic suture. This, in combination with the development of postoperative cuff cellulitis, could account for this unusual complication. Routine closure of the vaginal cuff at abdominal hysterectomy may decrease its chances of developing.

Treatment of tubal prolapse has ranged from simple cautery to laparotomy.[8] Simple cautery usually does not provide permanent cure; high ligation and excision of the prolapsed portion of the tube is required in most instances. This can be done through a vaginal incision. The vaginal defect is excised and closed. In a few cases, laparotomy is necessary because of associated pathologic conditions in the pelvis.

Summary

We have described a case of tubal prolapse following abdominal hysterectomy. It is usually observed after vaginal hysterectomy. Infection and hematoma formation appear to be the most important predisposing factors. This rare complication must be considered when a patient complains of abundant vaginal discharge in the few months following the operation and a polypoid mass is found at the vaginal vault. Biopsy is usually required for definitive diagnosis.

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Chemonucleolysis in Private Practice

STEPHEN E. NATELSON, M.D.

Introduction

The dissolution of ruptured lumbar disks with chymopapain was first attempted by Lyman Smith, M.D.[1] The drug was approved for investigational use in the United States by the FDA and abruptly withdrawn, as some neurosurgeons had serious misgivings about its efficacy and safety. A very careful study under the auspices of the FDA resulted in a positive recommendation in 1982 and in release of the drug for therapeutic use.[2] In spite of attempts by Smith Laboratories, the American Academy of Orthopedic Surgery, and the American Association of Neurological Surgeons to train and certify physicians in this procedure, there was an alarming number of instances in which the drug was apparently improperly administered, resulting in some severe neurological consequences, and even death.[3-5] Although experts in this field believed that these complications were related to operator error, there remained a lingering doubt about something inherently wrong with the drug itself, such that it constitutes a random, arbitrary hazard even when skillfully utilized. Among others, Fager,[6] a neurosurgeon, has vocally opposed the use of this drug in various professional fora.

I attended the course offered by the American Academy of Orthopedic Surgery and the American Association of Neurological Surgeons and commenced performing chemonucleolysis (IDC) in selected patients in March 1983, and by December 1985, believed I had accumulated sufficient data to permit a review of the efficacy and hazards of IDC to provide guidance for treatment of ruptured disks.

Method

I reviewed the charts of all patients whom I had treated by intradiskal injection of chymopapain between March 17, 1983 and Dec. 20, 1984. Each patient had exhausted conservative therapy and would normally have come to open operation. A choice of procedures was offered, and the patients chose IDC. The minimum follow-up available was 18 months and the maximum more than three years. Success was measured by returning to previous employment full time, or to normal daily activities. It should be emphasized that success does not mean being totally symptom free. General anesthesia was utilized throughout the first half of these cases by hospital edict, and local anesthesia was used thereafter when hospital policies changed. All procedures were done in the operating room. All patients were treated preoperatively for at least 24 hours with oral cimetadine (Tagamet) and benadryl and were given intravenous cimetadine, benadryl, steroids, and a fluid load immediately prior to the procedure. The product used in all cases was Chymodiactin (Smith Laboratories), usually 2 cc, though in a few patients toward the end of this period only 1.5 cc was given if the patient began to complain of excessive pain while awake upon reaching that dose. A skin test[7] became available during the last half of the study period, and was utilized thereafter; during the first half of the study, no allergy testing of any kind was carried out.

Accumulated Data

There were 131 patients, of which 84 were male and 47 female; the average age was 38 years (range 21 to 89). Intraoperative diskography was routinely carried out in all but 37 patients, most of whom had preoperative diskography. IDC was carried out at L4-5 50 times, with no diskogram in 22 of the patients, and at L5-S1 45 times, without diskogram in 10. Two separate levels were injected 25 times twice without diskogram; it is likely that many of the patients injected at two levels would have done just as well with one. Under tight guidelines, chymopapain can never be injected on the same patient at a second sitting, and in these patients the second disk was quite bulging on myelogram or CT or both, which explains the relatively high percentage of two-level injections. Nine patients were injected at L3-4 (three had no diskogram), and one patient

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each at L2-3 and D12-L1; both of these patients had diskography. There were 26 failures in 131 patients, indicating a success rate of 80%; there was no case of anaphylaxis. Success rates by level were L4-5, 41 of 50 (82%), and L5-S1 35 of 45 (78%). Surprisingly, 20 of 25 patients injected at two levels recovered (80%). Of ten patients with previous surgery who were injected, six experienced relief of symptoms sufficient to return to work without further treatment, indicating a success rate of 60%.

Analysis of Failures

Of the 26 failures, 21 were male (25%) and five female (16%); six of the 26 failed to be improved by subsequent surgery. At surgery, no ruptured disk, facet syndrome, or other pathology was clearly demonstrated. Long-term involvement with this group of patients suggests that they have serious psychological and compensation problems, and probably would not have improved with any treatment. On review, the preoperative studies showed a clear ruptured disk in all six.

A free fragment was found at surgery in seven of 26 patients. One of these at D12-L1 had previously been operated upon elsewhere; a policeman, he was able to return to his normal occupation, as were all of those with free fragments. Two of these free fragment patients had been injected at two levels, three at L5-S1 and one at L4-5.

The facet syndrome was identified at surgery in six of 26 patients; the disk was not found to be ruptured when exposed. All returned to normal activity. One unoperated patient developed opposite sciatica and his CT scan seems to indicate a facet syndrome on the opposite side following IDC. This case is involved in compensation litigation, and he has refused further intervention of any kind, nor is he working. Three of 26 patients were found at surgery to have both a tight facet and a soft bulging disk with a thickened ligamentum flavum. A combination of factors was thought to be responsible for their treatment failure; the disk was removed and the facet was decompressed, and all three recovered. One patient with spondylolisthesis and a ruptured disk was subjected to IDC on advice of an orthopedic colleague, though with personal misgivings. The

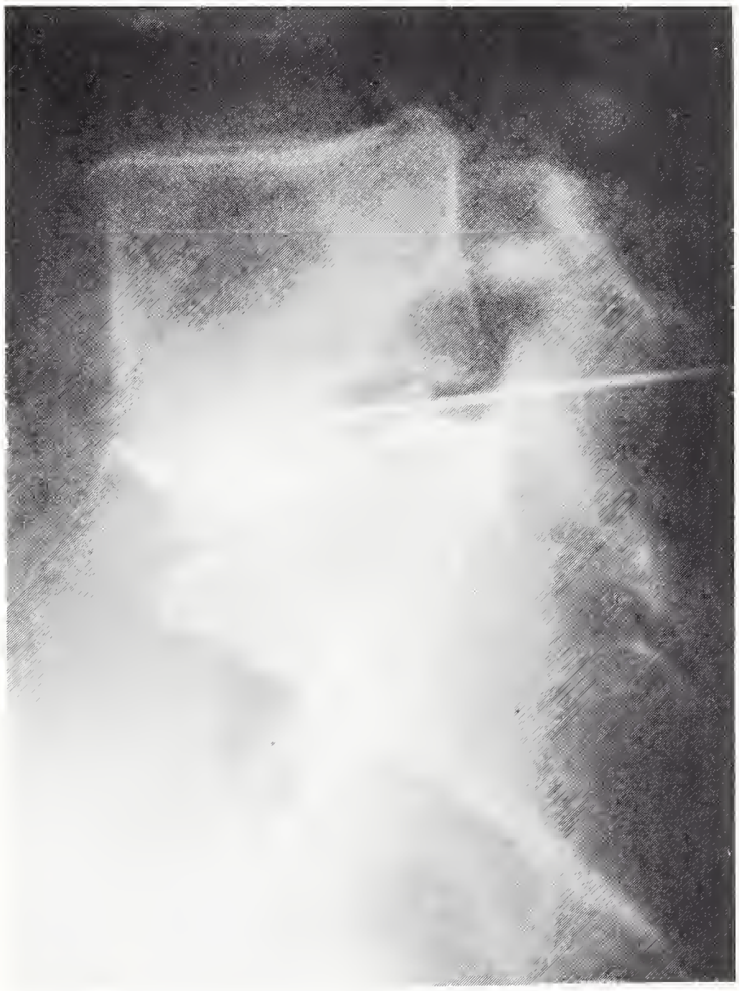

Figure 1. Diskogram of patient showing extravasation of contrast material at L5-S1 indicating ruptured disk. Note the normal cortical bone margins.

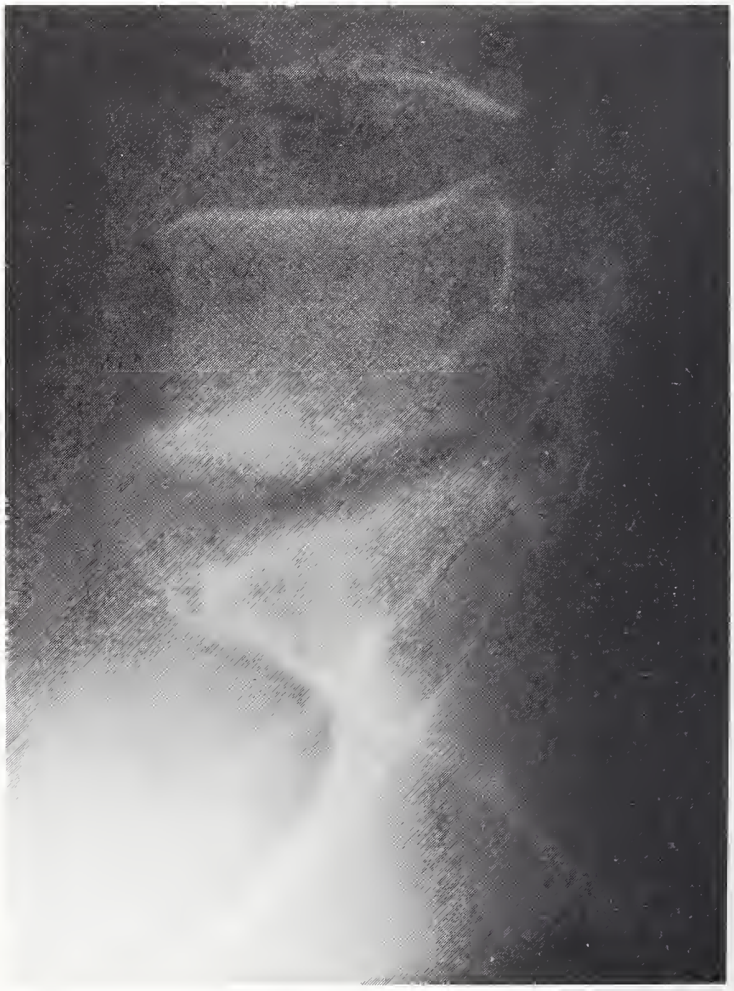

Figure 2. Same patient as Fig. 1, 18 months later, demonstrating effects of aseptic diskitis.

patient's pain became worse if anything, and he came to Gill procedure and fusion; no ruptured disk was found, and he is now recovered.

The radiographs of the one patient who developed aseptic diskitis are shown preoperatively and postoperatively in Figs. 1 and 2. The patient's radicular pain disappeared, but he had a prolonged period of backache; though he is now comfortable at rest, he is unable to do any physical labor and is not qualified by education for any other employment. He is not back at work.

One frankly psychotic patient with a large ruptured disk was treated with the concurrence of his psychiatrist. He complained bitterly preoperatively and postoperatively, and it was difficult to assess our results. Though he subsequently moved to another state, telephone inquiry recently revealed that he has had "two surgeries" and considers himself completely disabled.

It would thus appear that by this plan of treatment, we are left with a residual of nine patients out of 131, or 6% of the original group, who may be considered treatment failures.

The radiologic findings of all 26 treatment failures were critically reviewed; in only nine of 26 could anything noteworthy be recognized. Five patients had an extremely large ruptured disk. Total extrusion could not be definitely diagnosed from the myelogram or CT except by size. Two additional ruptured disks were foraminal; although some foraminal disks were included in the successes, this is worth mentioning. Three patients retrospectively had some degree of foraminal stenosis prior to IDC. One of the patients mentioned above with a very large ruptured disk simultaneously had mild foraminal stenosis.

Discussion

Although the following statements cannot be subjected to statistical defense from this series, it would appear that patients with very large ruptured disks should probably have open surgery and perhaps patients with foraminal disks also. Foraminal stenosis should be carefully identified and avoided in the performance of this procedure. Patients with spondylolisthesis should have open surgery. It should be realized, nonetheless, that some patients with these relative contraindications have excellent results. Patients with L5-S1 disk ruptures do less well than those at L4-5, possibly because either more ruptures at L5-S1 are extruded or foraminal stenosis tends to be more prevalent at L5-S1, or both.

That women did better than men in this group may be related to socioeconomic factors. The women had fewer workmen's compensation claims and less requirement for strenuous physical activity as part of their normal daily routine.

The ideal patient for IDC suggested by these results would be a woman who has a moderate-sized ruptured disk that is not foraminal at L4-5, and who has no evidence of spinal stenosis.

The reported dangers of this procedure have not been confirmed in this series of cases, and the procedure appears to be effective when used either selectively or in combination with surgery for failures. A failure rate of 6% by this sequence of treatment is low according to most studies. Recent verbal communications by colleagues suggest that lower dosages of chymopapain may be equally successful, with less hazard and less postoperative pain.

Subsequent to this series, I have been using 1 cc of chymopapain in each interspace, and some practitioners claim to be using 0.5 cc with apparent success. With smaller doses of chymopapain, it is essential that the needle be perfectly positioned. It seems most reasonable that a small amount of contrast material introduced immediately prior to the chymopapain is the best guarantee that the needle is properly positioned, and that the injection is not intravascular[8] or intrathecal.[9] The new myelographic contrast agent, Isovue, may provide the greatest margin of safety of any contrast agent yet available in the United States.

The important scientific fact is that disk ruptures are demonstrably dissolved by chymopapain.[10] Skill and experience should produce a notable improvement in safety.

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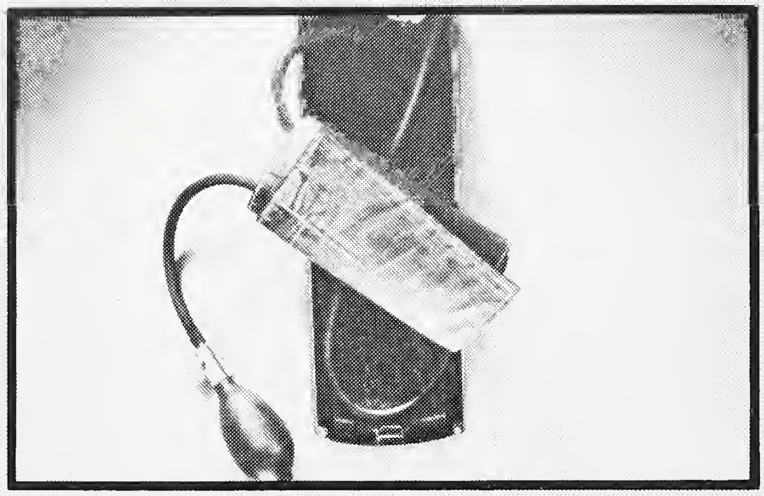

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Trauma Rounds

Rupture of the Azygos Vein: An Unusual Cause of Hemothorax Due to Blunt Trauma

GRIFFIN R. COATES, M.D. and DAVID P. HALL, M.D.

Introduction

Man's fascination with high speed travel has produced a steady increase in accidents on our streets and highways. Traffic fatalities are characterized by multiple injuries, and between 35% and 40% of these include chest injuries. In addition, the chest trauma can be the sole reason for the patient's demise or can often contribute significantly along with other injuries to an increased morbidity and mortality. Although the majority of patients with blunt chest trauma are in serious condition on arrival in the emergency room and in need of immediate and aggressive care, only approximately 10% to 15% of these require early thoracotomy. The indications for early thoracotomy after blunt trauma have been well described. We present an unusual case of massive hemothorax and review guidelines for the management of these injuries.

Case Presentation

A 63-year-old white woman was brought to the emergency room after being involved in a motor vehicle accident. The history obtained from the paramedics was that she had been wearing her seat belt, and there was extensive damage to her vehicle. It required approxiamtely 30 minutes to extricate her from the car, and her only obtainable blood pressure at the scene was 80 mm Hg systolic. On arrival in the emergency department, the patient complained of difficulty breathing and of right chest pain. Vital signs were pulse 120/min, blood pressure 80 mm Hg systolic, respirations 24/min and shallow; the trachea was in the midline. There were decreased breath sounds on the right side with dullness to percussion, multiple right rib fractures, and a flail segment in the right chest wall. The abdomen was unremarkable except over the suprapubic area, where it was tender to palpation. The MAST trousers were in place, so that the lower extremities could not be immediately examined. A chest x-ray confirmed multiple rib fractures on the right, as well as deviation of the mediastinum to the left due to a hemothorax. A sterile 14 gauge needle introduced through the anterior fourth intercostal space for decompression yielded blood, and a 36 French thoracostomy tube introduced into the right fifth intercostal space in the midaxillary line promptly returned a large amount of blood, approximately 700 cc over the next ten minutes. At this point the tube was clamped, and uncross-matched, O-negative blood was rapidly transfused into the patient; a right subclavian IV attempt was unsuccessful due to a large hematoma from a right clavicle fracture. With the transfusion, the patient's blood pressure increased to 120 mm Hg systolic. The MAST trousers were released slowly, and a complete examination was performed. A Foley catheter returned grossly bloody urine. As the patient's blood pressure stabilized, the chest tube was unclamped, and over the next 15 minutes an additional 800 cc of blood was removed from the right chest.

The patient was transported to the operating room, where a Cordis introducer was inserted and a right thoracotomy performed through the fifth intercostal space. The chest wall musculature had a large hematoma, and upon entering the pleural space, a massive clotted hemothorax was found and removed, using the autotransfuser. The lung parenchyma and hilar structures appeared to be normal, without bleeding or air leak, and there was no significant bleeding from intercostal vessels. A 10- to 12-mm tear was found in the superior vena cava at the junction of the azygos vein; this was rapidly controlled with pressure and oversewn using running 4-0 Prolene suture, stopping the major blood loss. The patient's blood volume was restored before proceeding with the exploration of the area and ligation of the azygos vein proximally, several intercostal arteries, and a bronchial artery. The tear in the vena cava was further repaired with interrupted mattress sutures; the mediastinal space over the superior vena cava, which was massive hemothorax, was not closed. The pericardium appeared normal, without evidence of tamponade, and the aorta was normal to palpation and inspection. Closer inspection of the lung and pulmonary vessels showed no evidence of compression injury or tear. After chest tubes were placed, the thoracotomy closed. Cystograms confirmed an extraperitoneal bladder tear, but an exploratory laparotomy was negative except for the pelvic hematoma and bladder injury. An IVP confirmed bilateral renal function and no evidence of renal injury.

Postoperatively, the patient had a coagulopathy, which was treated with fresh-frozen plasma and vitamin K replacement. A postoperative seizure was found on CT scan to be due to an intracerebral hematoma; her neurological status slowly improved and the intracerebral hematoma eventually resolved. Mechanical ventilation was continued for four days postoperatively, and chest tubes were removed on the fifth postoperative day. Her postoperative course was also complicated by hyperbilirubinemia with some evidence of acalculous cholecystitis by HIDA scan.

Discussion

Hemothorax is a common sequel to blunt chest trauma, with an incidence after a nonpenetrating chest trauma of between 25% and 75%. The

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From the Department of Surgery, Chattanooga Unit, University of Tennessee College of Medicine.

source of bleeding may be the lung, heart, great vessels and their branches, an intercostal artery or vein, mediastinal veins, or vessels of the diaphragm and chest wall. The majority of these injuries can be treated with a tube thoracostomy alone. If the hemothorax is large, the hypovolemia will cause hypotension and a decreased cardiac output, leading to acidosis; the large volume of blood in the pleural space can also cause a decrease in ventilation with hypoxemia.[1]

The physical signs and symptoms associated with hemothorax vary with the quantity of blood in the pleural space. If small amounts (less than 500 cc) are present, there may be little or no change in the patient's appearance or vital signs. With larger fluid losses (over 1,000 cc), there are the usual findings of internal hemorrhage, including pallor, shock, hypotension, restlessness, and tachycardia, as well as dyspnea and decreased or absent breath sounds. Chest x-rays are useful in demonstrating hemothorax of more than 150 or 200 cc. Although a hemothorax can be managed with repeated thoracentesis, the recommended management usually includes insertion of a large-bore thoracostomy tube, which allows both expansion of the lung and monitoring for continued bleeding. The insertion of a chest tube solely for hemothorax should be done during the initial assessment of the trauma patient; if only a small hemothorax is present, the chest tube can be inserted later following a complete evaluation of the patient's other injuries, since the removal of a small hemothorax early in the course of the patient's care may waste time without producing significant benefit. If the hemothorax is estimated on chest x-ray to contain over 1,000 cc, the insertion of the chest tube in the emergency room may cause exsanguination through release of the tamponade effect.

Since most causes of such a large hemothorax will require operative intervention, placement of the chest tube may be withheld until the patient is in the operating room and prepared for a possible thoracotomy if a large hemothorax is found; this minimizes the time between insertion of the tube and definitive treatment and avoids possible hypotension in the emergency room.

Indications for early surgery in blunt chest trauma include massive and persistent bleeding, acute cardiac tamponade, aortic rupture, injury to the supra-aortic branches, rupture of the trachea and bronchi, rupture of the diaphragm, and rupture of the esophagus.[2] The usual cause of massive and persistent bleeding is injuries to the internal mammary artery, intercostal arteries, great vessels, or heart. It is very important to rapidly restore the patient's blood volume when a massive hemothorax is encountered, since the insertion of the chest tube may release a tamponade effect on the bleeding site. In general, surgical intervention is indicated when the blood loss through the chest tube continues to exceed 500 cc/hr after three hours, or more than 150 cc/hr after six hours.[1] Obviously, any patient with continued hypotension should be considered for early operative intervention.

The association of severe thoracic and abdominal injuries associated with lap-harness seat belts has been previously elucidated,[3] and although these devices have undoubtedly reduced major injury from motor vehicle accidents, the documentation of their use cannot completely rule out significant injury in any given situation; in fact, the lack of facial and extremity trauma may make the initial impression of such patients deceptively benign.

Blunt chest injury is often associated with vascular injury, and the presence of a widened mediastinum or hemothorax usually indicates injury to the aorta or other major arterial branches. It should be remembered that venous bleeding can cause significant mediastinal hematoma and widening of the mediastinum on chest x-ray. The occurrence of azygos vein injury due to blunt trauma has been reported by Baldwin,[4] Salizzoni,[5] and Spagliardi.[6] It is important to remember that as shown by Baldwin[4] associated vascular injuries may also be present in the thorax, and such patients should be considered for angiography as soon as they are stable.

Summary

We have presented an unusual case of massive hemothorax from an azygos vein injury, and have briefly reviewed the indications for early operative intervention in blunt chest injury. The physician must be alert for possible associated injuries; the use of a seat belt or lap-harness cannot rule out such a possibility.

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Hemothorax

Case Reports

Case 1. An 82-year-old black woman with a long history of poorly controlled hypertension was transferred to Vanderbilt University Hospital for the evaluation of hypotension and chest pain. Her past medical history indicated accelerated hypertension with left ventricular hypertrophy, but she reported good health until the day prior to admission when left flank pain, nausea, and vomiting began. Six hours prior to admission she began to complain of left-sided pleuritic chest pain and a hacking nonproductive cough. When seen at her local hospital her systolic blood pressure was 90 to 100 mm Hg, for which she was treated with intravenous fluids and transferred to VUH. Admission examination showed orthostatic hypotension, tachycardia, and cool dry skin. Neurologic examination was normal with the exception of lethargy. Cardiac examination was normal. The chest was dull to percussion at the left base, with decreased breath sounds.

Laboratory examination showed a white blood cell count (WBC) of 23,700/cu mm with a left shift on differential, a hematocrit of 28.7%, and a platelet count of 240,000/cu mm. Coagulation studies and electrocardiogram were unremarkable. Chest x-ray revealed a left pleural effusion (Fig. 1), and when compared to previous chest radiograph nine months earlier (Fig. 2) dramatic widening of the mediastinum was apparent. A thoracentesis yielded bloody fluid with a hematocrit of 17%. Repeat complete blood cell count (CBC) showed a hematocrit in the peripheral blood of 17%.

A left chest tube was placed and immediately drained 600 cc of blood. Emergency aortography demonstrated a type III thoraco-abdominal aortic aneurysm starting distal to the left subclavian artery and extending to the aortoiliac bifurcation. The celiac, superior and interior mesenteric and renal arteries were uninvolved. The patient was treated with replacement of fluids, packed red blood cells, and combination of beta blockers and nitroprusside for blood pressure control. The chest tube was removed without further bleeding and she went on to have an uneventful recovery and eventual hospital discharge.

Case 2. A 69-year-old white woman was admitted for evaluation of confusion and a left pleural effusion. The patient was in good health with the exception of moderate hypertension and adult onset diabetes mellitus controlled with an oral hypoglycemic agent. Two recent episodes of hypoglycemia had prompted a reduction in the dose of the hypoglycemic agent.

On the day of admission, she was found lying on the floor of her home, confused and complaining of dull substernal chest pain. On arrival in the emergency room, she had a blood pressure of 146/70 when supine, with a 30 mm Hg drop on

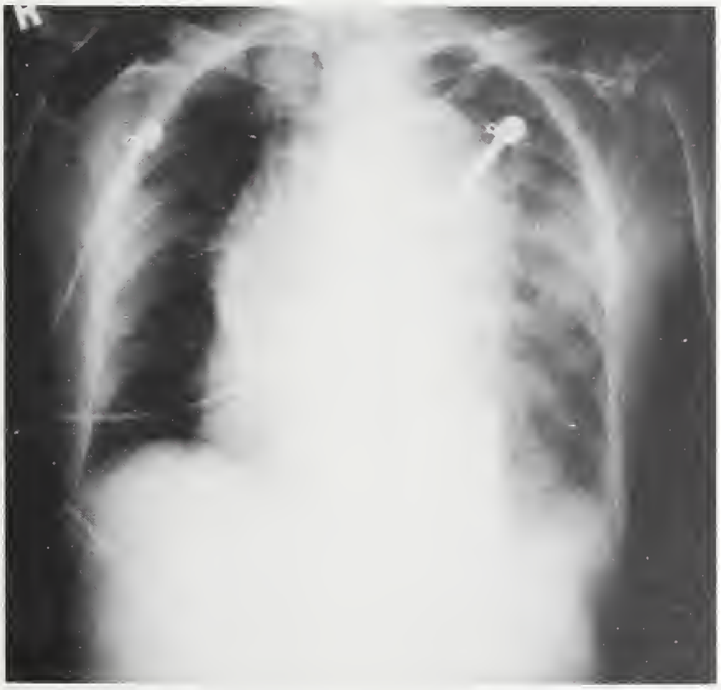

Figure 1. Chest x-ray demonstrating widened mediastinum and left pleural effusion.

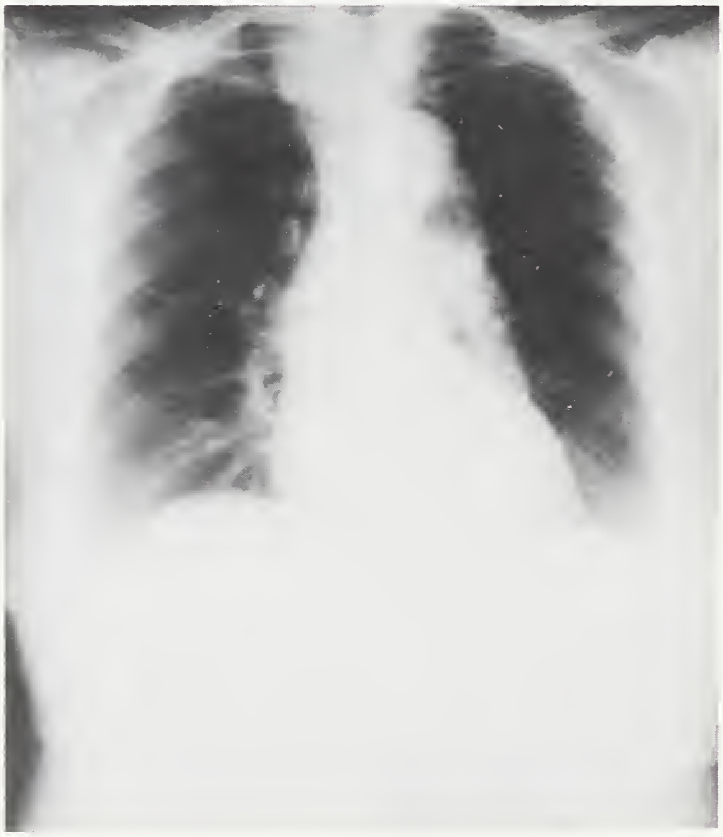

Figure 2. (Case 1) Chest x-ray taken nine months before Fig. 1, demonstrating normal mediastinum.

Prepared by Arthur P. Wheeler, M.D., Hugh J. Morgan chief medical resident, Vanderbilt Medical Center, Nashville.

sitting. She was afebrile, and had a regular pulse at a rate of 90/min. She had an ecchymotic area over the left hip. Cardiac examination revealed a normal S1 and S2 with a grade 2/6 systolic ejection murmur. The left chest was tender to palpation, with dullness and absent breath sounds over the lower half of the lung field. Neurologic examination was normal.

Laboratory examination showed a serum sodium of 120 mEq/liter and a normal serum glucose. CBC revealed a hematocrit of 26.2% and a WBC of 9,200/cu mm with a normal differential. Electrocardiogram was normal. Chest radiograph initially was interpreted as showing a calcified aortic knob and a large left pleural effusion. Thoracentesis yielded bloody fluid with a hematocrit of 17%.

Because of the suspicion of a leaking aortic aneurysm, emergency aortography was obtained, but it proved to be normal. Under fluoroscopic examination, fractures of the left posterior third and fourth ribs were suspected; this was confirmed by repeated chest x-ray (Fig. 3). Treatment included intravenous fluid and blood replacement and placement of left chest tube that drained approximately 1,500 cc of fluid and blood. The patient had an uneventful hospital stay, recovering completely prior to discharge.

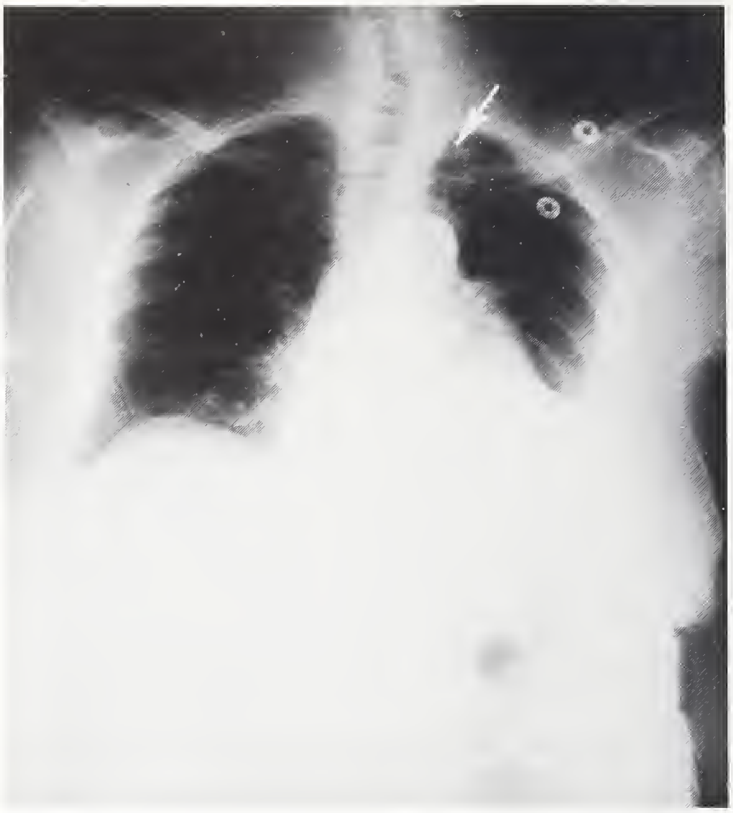

Figure 3. (Case 2) Chest x-ray demonstrating left pleural effusion and fractured posterior rib (arrow).

Discussion

A hemothorax is a collection of bloody fluid in the pleural space having a hematocrit of 50% or more of that of circulating blood. Since a hematocrit as low as 5% in pleural fluid can appear grossly bloody, hematocrit should always be determined on bloody pleural fluid. While in the vast majority of cases hemothorax is secondary to blunt or penetrating trauma,[1,2] as in case 2, a variety of unusual cases have been described. Bleeding may complicate any invasive procedure of the thorax, including central venous catheter placement, pleural biopsy, or thoracentesis. The rupture of thoracic vessels, as in case 1, is an uncommon cause. Case 1 also demonstrates that even dissecting aneurysms may be conservatively managed. Rupture of aortic aneurysms, splenic artery aneurysms, pulmonary arteriovenous malformations, and patent ductus malformations have all been reported as producing hemothoraces.[3,4]

Spontaneous bleeding into the pleural space has been seen in conditions with altered hemostasis, such as anticoagulation therapy, hemophilia, and thrombocytopenia. In most cases, "spontaneous" hemothorax follows pulmonary thromboembolism with or without pulmonary infarction.[5] The treatment of "spontaneous" hemothorax requires discontinuation of anticoagulant therapy and replacement of deficient clotting factors or platelets.

While therapy of hemothorax remains controversial,[2] prompt evacuation of blood from the pleural space is the mainstay of therapy. Complete drainage of the pleural space will prevent the most frequent complications of hemothorax, which are empyema, fibrothorax, and chronic pleural effusion. Most practitioners favor initial closed thoracostomy drainage with a large bore (36 or 40 French) chest tube, followed by open thoracotomy only for continued massive hemorrhage or residual undrained blood. Overall, approximately 20% of patients with hemothorax will require thoracotomy for control of bleeding or evacuation of pleural hematoma.

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MRI Case of the Month

MICHAEL C. GAROVICH, M.D.; CHARLES L. ROBINETTE, JR., M.D.; and
EVERETTE I. HOWELL, M.D.

History

A 63-year-old white man complained of dizziness, accompanied by vertigo and one episode of sixth nerve palsy; CT scan one year earlier had revealed enlarged ventricles, again shown on a repeat scan two months later. An arteriogram was nonrevealing. The magnetic resonance imaging (MRI) scan is presented. Please evaluate Figs. 1 and 2 and see if you can make the correct diagnosis.

Discussion

MRI has revolutionized central nervous system (CNS) imaging in a way similar to the revolution that occurred with CT. MRI of the CNS has higher inherent tissue contrast than CT, lacks bone artifacts, requires no ionizing radiation or intravenous contrast injection, and can image in any plane. Its disadvantages are lower spatial resolution and poor sensitivity in detecting intracranial calcifications.

This case illustrates superiority of MRI in demonstrating a lesion at the foramen magnum and its effect on the brainstem and cord. There is no bone artifact, and no intravenous contrast was used.

DIAGNOSIS: Clivus meningioma (mixed type). The tumor was resected using a microscope under high magnification with the aid of a laser.

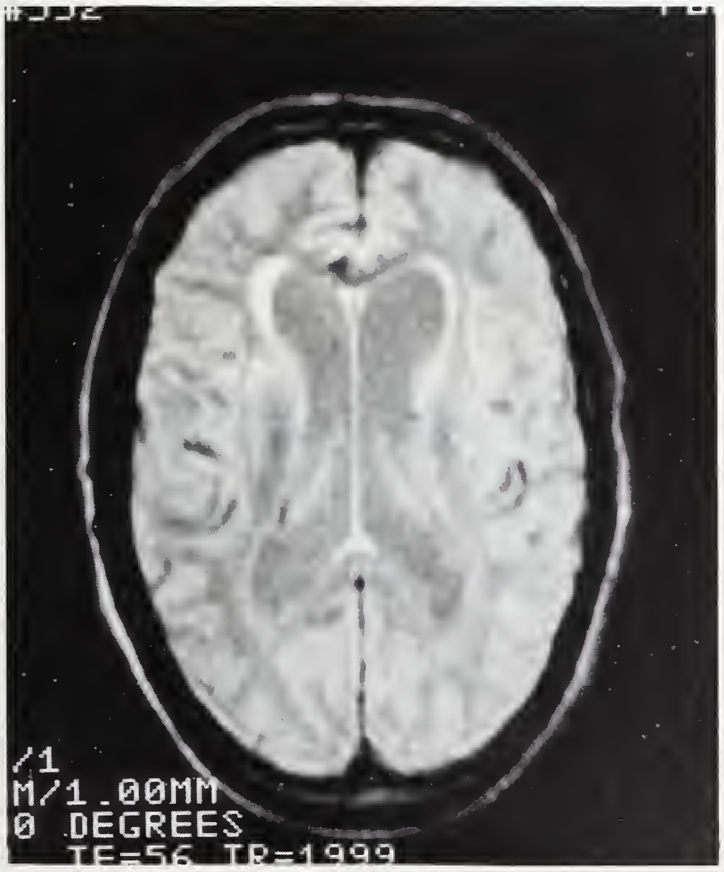

Figure 1. The lateral ventricles are enlarged, with periventricular hyperintensity thought to represent transependymal resorption of CSF. T_2 weighted image.

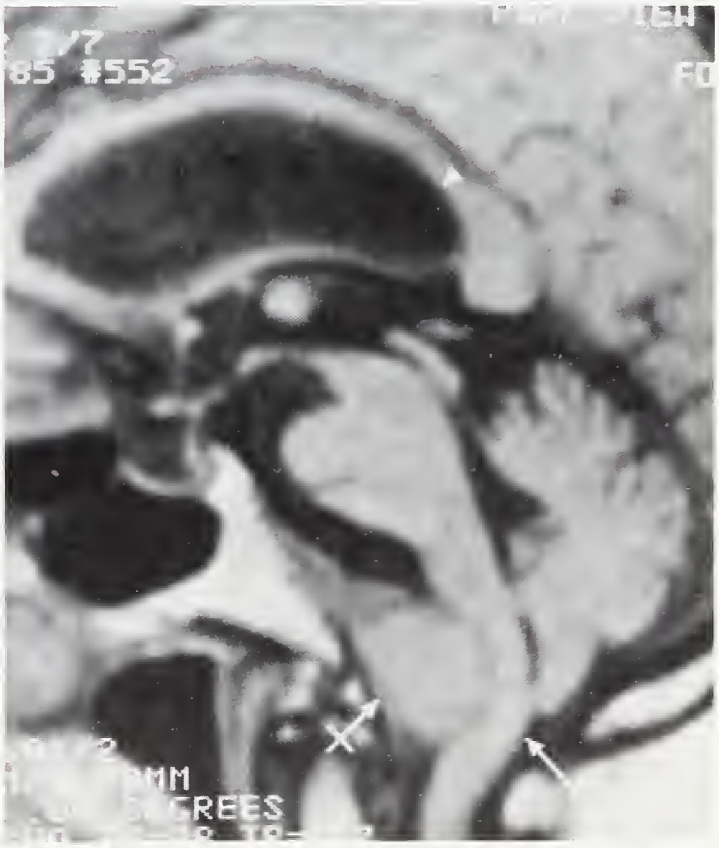

Figure 2. Sagittal T_1 weighted image demonstrates the enlarged lateral ventricle (arrowhead), third ventricle, and fourth ventricle, as well as the aqueduct of Slyvius. The medulla and upper cervical cord are compressed (arrow) by the clivus meningioma (crossed arrow).

From the Departments of Radiology (Drs. Garovich and Robinette) and Neurosurgery (Dr. Howell), HCA Park View Medical Center, Nashville.

Radiology Case of the Month

A Patient With Carman-Kirklin Sign on a Barium Study

P. H. PATEL, M.D.; SANKAR LAKSHMAN, M.D.; and EAPEN THOMAS, M.D.

Case Report

A 66-year-old white man had a history of diffuse, dull-aching abdominal pain of three months' duration, associated with anorexia and postprandial abdominal bloating. The pain was not relieved by antacids, and was unaccompanied by nausea, vomiting, hematemesis or melena. He denied alcohol or tobacco abuse and did not use coffee. He had a peptic ulcer treated with gastrectomy 35 years earlier. The physical examination was essentially unremarkable. Laboratory data revealed normocytic anemia and abnormal liver function tests with alkaline phosphatase higher than 350 U/liter. An upper gastrointestinal barium study is shown in Fig. 1. What is your diagnosis?

(1) Gastric outlet obstruction
(2) Anastomotic ulcer
(3) Gastric cancer
(4) Normal barium study

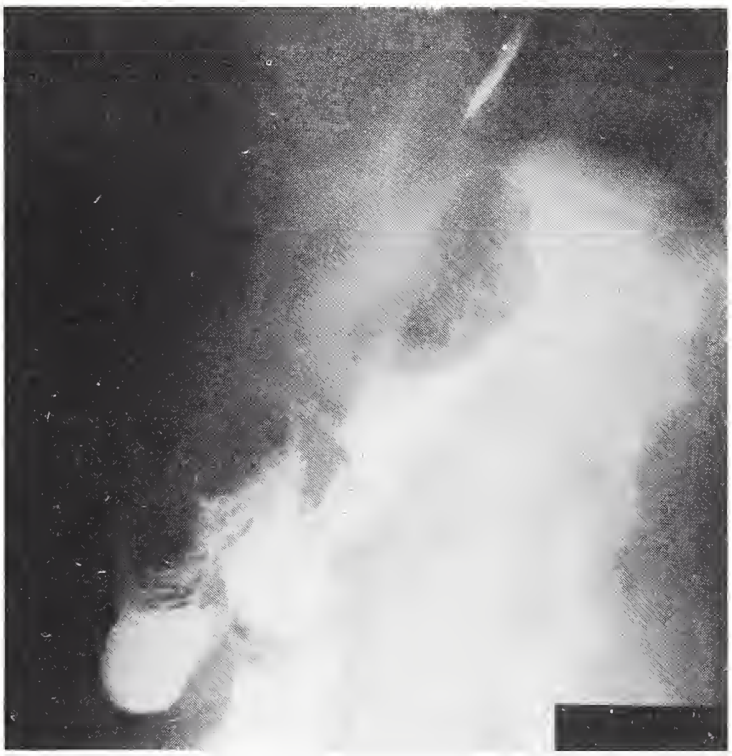

Figure 1. Upper gastrointestinal barium study.

The barium study shows evidence of a Billroth-I gastrectomy. The mucosa of the distal gastric stump is abnormal with a "shelf" like appearance anteriorly (Fig. 1), and the compression spot film of the gastric stump shows Carman's meniscus sign (Fig. 2). When viewed in profile, the combination of an intraluminal crater surrounded by a nodular halo forms Carman's meniscus sign or complex, first defined by Carman and later elaborated by Kirklin. The ulcer in this

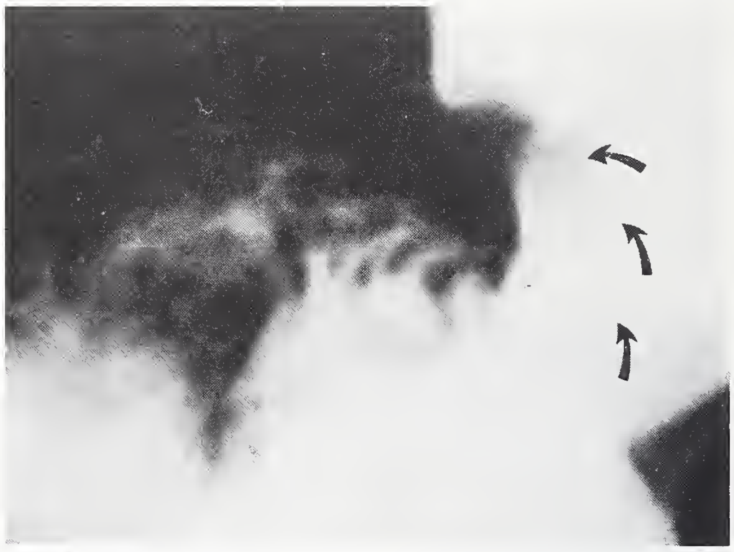

Figure 2. A compression spot film revealing the Carman's meniscus sign (arrows).

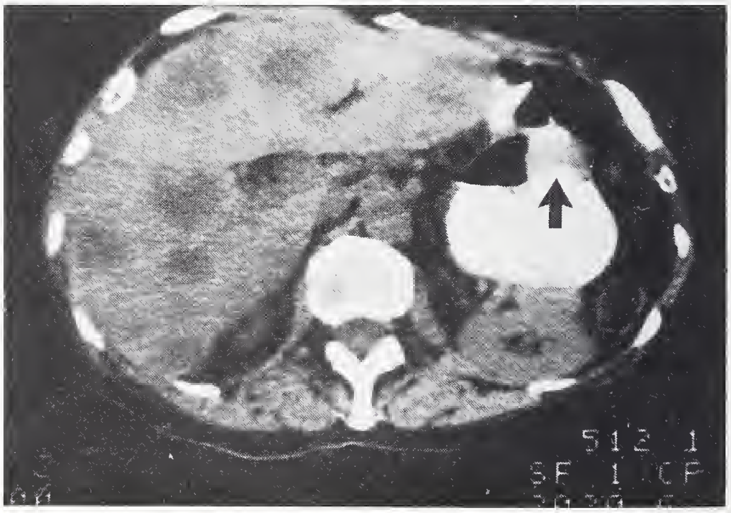

Figure 3. CT scan of the abdomen. The mass in the stomach is seen close to the anastomotic site (arrow). There are multiple areas of low attenuation in the liver, representing metastatic disease.

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patient extends into the anastomotic area.

The CT scan of the adomen shows the mass lesion of the gastric stumpt to be very close to the anastomotic site (Fig. 3). Multiple areas of decreased attenuation within the liver represent metastatic disease.

Because the barium study was suspicious for a neoplastic lesion, an esophagogastroduodenoscopy was done. The esophagus was normal, but near the anastomosis, there was a large fungating mass with a central ulcer consistent with a gastric carcinoma and this was confirmed by multiple biopsies. The elevated alkaline phosphatase suggested liver metastases, and this was confirmed by the CT scan. This type of cancer is often referred to as gastric stump carcinoma.

Discussion

Carcinoma of the stomach was the most frequent cause of death from cancer 50 years ago. The incidence has declined since then.[1] Presently an estimated 25,000 cases occur annually in the United States. The etiology of the disease is still not clear. The disease continues to be a common killer in some countries like Japan, Chile, and Iceland. Thus, environmental factors may be important. Although difficult to study, diet has been implicated in the pathogenesis in several studies. Some families with gastric cancer have been described implicating genetic factors. Several conditions have been associated with a higher incidence of gastric carcinoma, including pernicious anemia, hypochlorhydria, achlorhydria, post-gastrectomy stomach, hypertrophic gastropathy, and gastric polyps; atrophic gastritis and intestinal metaplasia are considered to be the major precursors.[2]

The association of partial gastrectomy and subsequent development of stump carcinoma was first described by Balfour in 1922.[3] Subsequently, multiple studies have been done to evaluate this association. Results have frequently been conflicting. Studies in which comparison between the observed incidence of stump cancer has been made with the expected incidence of gastric cancer in a similar population have shown no significant differences between the two groups.[4] It has also been argued that the incidence of cancer in post-gastrectomy patients cannot be higher, because in these patients the most common site for cancer has been removed.[5] That there is no significant difference in the observed versus expected incidence of gastric cancer in some of the studies may indicate that the incidence in the remaining stomach is indeed higher. Animal studies have clearly demonstrated increased incidence of stump cancer. In addition, these studies have also shown that the incidence is very high when a gastroenterostomy is done without gastrectomy. When a Billroth-II gastrectomy is done with a Roux-en-Y gastroenterostomy in these animals, the incidence of gastric cancer approaches zero.[6] Thus procedures having more duodenogastric reflux carry higher risk; the risk also increases with the interval after the surgery.

It is believed that the risk of developing cancer in post-gastrectomy stomach is 3%, although in the first 15 years after the surgery it is minimal. Should these patients be routinely screened to diagnose early gastric cancer? It has been recommended that screening should be done by endoscopy with multiple biopsies, but only after 15 years. Screening should be done every three to five years unless the biopsy reveals dysplasia, when more frequent follow-up would be necessary.

Answer: (3) Gastric stump carcinoma.

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Health and Environment Report

The Status of the Nation and Tennessee in Reaching the 1990 Health Objectives

FREDIA S. WADLEY, M.D., MSHPA

Many feel the United States Public Health Service (USPHS) was exceedingly brave to make a commitment to the 226 health objectives that were published in *The 1990 Health Objectives for the Nation*. The USPHS probably deserves even more respect, however, for attempting to give us a status report by means of *A Midcourse Review: The 1990 Health Objectives for the Nation*.[1]

Though space does not permit discussion of all 226 objectives, I shall present the major components of some of the objectives related to the vaccine-preventable diseases and give a national and state progress report. Table 1 summarizes the objectives and achievements related to the annual number of reported cases of these diseases. Table 2 gives some of the goals relating to "Improvement of Health Services."

Most health professionals believe there is little chance of having 90% of 2-year-olds completely immunized by 1990 (Table 2). Tennessee counted only those children who had received all the recommended immunizations using four DPTs as the standard, while the United States used only three DPTs. The U.S. percent of 2-year-olds completely immunized by Tennessee's standards would probably not be higher than our 67.8%.This points out the difficulty in comparing figures that are obtained using different guidelines for data collection.

The two health service objectives relating to influenza and pneumococcal vaccine (Table 2) cannot be evaluated at this time in Tennessee because we have no reporting or surveillance system for them. It is not likely, however, that our progress would be any better than the nation's. It is obvious that neither health professionals nor

TABLE 1

1990 GOALS FOR IMPROVEMENT OF HEALTH STATUS

	1990 US Goals	1985 US Cases	Possibility of US Achievement by 1990	1985 TN Cases
Measles	500/yr	2,704	Questionable	2*
Mumps	<1,000/yr	2,886	Likely	32†
Polio	<10/yr	5‡		0
Rubella	<1,000/yr	604	Achieved	0
Congenital rubella syndrome	<10/yr	0	Achieved	0
Diphtheria	<50/yr	2	Achieved	0
Pertussis	<1,000/yr	3,275	Unlikely	18
Tetanus	<50/yr	71	Unlikely	12

*Imported case with second case in cousin who has delayed measles immunization until after Christmas.
†Outbreak of mumps in Tennessee in 1986 with total reported cases for year 250 but estimated to have over 600 cases.
‡All polio cases vaccine related.

From the Tennessee Department of Health and Environment, Nashville. Dr. Wadley is chief medical officer of TDHE.

TABLE 2

1990 GOALS FOR IMPROVEMENT OF HEALTH SERVICES

Goal	US Status	TN Status
	(1984 Data)	*(1986 Data)*
90% completed basic immunizations by age 2	measles–81.7% mumps–76.7% rubella–78.4% polio–74.2% DPT–85.8%	67.8% with all
The US figures are based on three DPT injections; Tennessee uses four DPTs.		
	(1984-85 School Year)	*(1985-86 School Year)*
95% in licensed day care and K-12 fully immunized	Head Start–93% Day Care–93% K-1–96% K-12–88%	98.6% 98.4% 98.9% 97.8%
60% of high risk population* receiving annual immunization against influenza	1984–17% Achievement unlikely	Unknown
60% of high risk population* received pneumococcal vaccine	1984–9.5%	Unknown

*As defined by the Immunization Practices Advisory Committee of the Public Health Service.

the public are convinced of the value of these vaccines; it could be that although we are convinced of the value of immunizations for children, we are not as accepting of them for adults.

Another service objective is that by 1990 at least 50% of individuals in populations designated as targets by the Advisory Committee on Immunization Practices should be immunized within the five years of licensure of a new vaccine for routine clinical use. Hepatitis B and *Haemophilus influenzae* b (Hib) vaccines have been licensed for routine clinical use since this objective was established. By 1985, about 20% of those targeted to receive hepatitis B vaccine were immunized, and 30% of targeted health professionals had received the vaccine. Hib was not licensed until April 1985, but by the end of 1985 an estimated 3 million doses had been given in the United States.

It is possible for this objective to be met, but a great deal of effort is still needed. The Centers for Disease Control in Atlanta is striving to improve professional and public awareness about the benefits and risks of hepatitis B vaccine. The greatest barrier for the use of the Hib vaccine is the lack of federal and state funds to provide this vaccine through local health departments. Tennessee is one of the few states purchasing the vaccine and requiring it for attendance at day care facilities; Tennessee gave 36,000 Hib injections in public health clinics alone in 1986. For these reasons, Tennessee should easily achieve this objective for Hib.

It appears that the greatest challenges for the United States and Tennessee are (1) to complete the basic recommended immunizations in children by age 2; and (2) to make health professionals and the public aware of the immunizations needed in certain high risk groups.

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Loss Prevention Case of the Month

Patient Care and Bureaucracy

J. KELLEY AVERY, M.D.

Case Report

An 84-year-old white female stroke victim was admitted to a long-term care facility from a leading Tennessee hospital with the following orders signed by the medical director, who became the patient's attending physician:

(1) 1,800-calorie ADA diet
(2) AC urine for glucose, one time daily, sequentially before breakfast, lunch, and supper
(3) Up in chair ad lib
(4) Decubitus care per standing orders b.i.d.
(5) 15 units NPH insulin q a.m., AC

There was no note in the hospital chart at the time of discharge describing any decubitus lesions. The entry history and physical examination in the nursing home chart did not mention decubitus ulcers. Six weeks after admission a progress note on the record indicated no change in the patient's condition, but an order for "C&S on decubitus ulcers" was written.

During the following 3½ months, there were orders for different antibiotics on three separate occasions, the first of which was culture specific. The first antibiotic order was for Ampicillin, 250 mg, PO, q.i.d., for 10 days. No subsequent cultures were done for the changes in antibiotic therapy. On one occasion an order was written for a topical antibiotic to be applied "to the decubitus ulcers on the inner aspects of each leg." There were recertification forms for Medicaid signed at the required 60-day intervals, but no progress notes in the patient's medical record.

Six months after admission to the nursing home, the patient was readmitted to the general hospital where bilateral above-knee amputations were done. The patient died approximately 15 days after this hospital admission. A lawsuit was filed within a year of the patient's death charging both the nursing home and the medical director with negligence.

Loss Prevention Points

A careful study of the patient's medical records in the nursing home and in the hospital revealed unmistakable evidence of advanced arteriosclerotic disease involving both the brain and the lower extremities. There were notations in the nursing home record indicating repeated telephone contacts with the attending physician, but only one physician visit could be documented. The tests for urine sugar were done with some regularity and ranged from 0 to 3+. No blood glucose levels were done. While the nurses' notes faithfully record "patient turned q two hours," there was no evidence that the "up in chair ad lib" order was ever adhered to.

This elderly diabetic patient obviously succumbed to the ravages of generalized arteriosclerotic disease. Even though in all probability the course of this disease could not have been significantly altered by more aggressive therapy, a sizable settlement was agreed to by both the nursing home and the attending physician.

According to experts who were involved in the review of the records in this case, the attending physician did not adequately supervise the patient's care while she was in the nursing home. Experts who reviewed the record agreed that decubitus care was substandard, and that the diabetes was not monitored adequately. The "decubitus lesions" were never described or even precisely located anatomically in the record. Although the attending physician was "medical director" of the facility, the record did not show that the physician had been made aware of changes in the patient's condition in a timely manner.

The record did show that the system worked well enough to satisfy the bureaucratic requirements of recertification for Medicaid payment, but it did not reflect a comparable level of interest in the clinical management of the patient.

The number of patients who will be cared for in this type of setting can be expected to increase as our population gets older. The medical records must show the physician's direct supervision of patient care as *clinically* indicated. We must not allow any type of "regulation" to dictate the level of involvement in the care of our patients in long-term care facilities.

Dr. Avery is medical director for Continuing Medical Education, St. Thomas Hospital, Nashville, and chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood.

James R. Royal

Our Time

Have you ever asked yourself, "What is time?" It's a word we use very frequently and in dozens of ways with various connotations. The early Greeks had a dual concept of time and it is reflected in the early translations of the Bible and other ancient writings.

They had two primary words for time. "Chronos," from which our word chronometer is derived, was meant to designate measured time—defined as "a continuous, irreversible occurrence of a succession of events from the past through the present to the future." Seconds, minutes, hours, days, weeks, years, decades, centuries, and on to millenia were adopted to measure the shadow on the sundial, the running of the sand through the hourglass, the striking of the clock, the calendar with day and night, summer and winter. This is the time we live by, work by, and die by. It passes unrelentlessly. How we use that time is our life and determines our impact on human history. As physicians we have more opportunities to spend our allotted time for the good of our patients, communities, and country than any other profession.

Henry Van Dyke wrote "*Time Is*: Too slow for those who wait; Too swift for those who fear; Too long for those who grieve; Too short for those who rejoice; But for those who love, Time is eternity." Of all the things we can do with our time, such as use, waste, kill, bide, spend, buy, lose, gain, save, keep, give, and dozens of others, use is probably the most significant.

The other concept of time, the Greek word "kairos," implies an age, a season, a definite period, or an opportunity. It may be the time for action, to make things happen, to change, or even the old "come to the aid of the party." It is this time that is upon us now if we are to have an impact on the future of health care in Tennessee, U.S. of A.

Changes are being forced upon us—some good, some bad. The remarkable advances in medicine are promising longer, healthier lives for all of us. The economics and ethics of delivering these to our patients are being debated and settled in Congress and state houses across the nation. Insurance companies, hospitals, employers, and other third parties are staking out their turfs, and all overlap onto the practice of medicine.

It's TMA's time, it's AMA's time, it's medicine's time to become involved in the solutions of today's problems. Fate does not hold the future. We do. It's our time.

James R Royal, MD

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MARCH, 1987

editorials

The Face on the Bottle

> So we sing of Lydia Pinkham,
> Savior of the human race:
> She gave the world her vegetable compound—
> And on each bottle was a picture of her face.
>
> —Anon.

Many of you will recognize that as the chorus of a bawdy song we used to sing in my medical school and housestaff days. Maybe they still do, though I get the impression students don't have as much fun—or perhaps better phrased, they don't engage in the same level of extracurricular frivolity—as we did. In the first place, most of the students now are married, whereas in those days a married student was an anomaly. In the second place, the students are loaded down with all sorts of nonmedical trivia—necessary, perhaps, but nonetheless trivia—having to do with our socioeconomic problems, which are slowly but surely choking medical practice to death—or at least squeezing all the fun out of it.

In the frontier days of our country, medicine was often a sure enough sideshow. Anyone was a doctor who said he was, and the only limits on so-called medical practice was buyer credulity. Legislatures regarded as self-serving the attempts by the more responsible members of the medical community to define criteria and establish licensure for the practice of medicine. Though the term had not yet been coined, licensure was considered in restraint of trade. Much of medicine was indeed a trade in those days, and its motto was "Caveat emptor." Lydia E. Pinkham and colleagues were in the driver's seat.

Professionalism was established in medicine in this country shortly before the end of the last century, as one state after another, detecting the wisdom in the advice of medicine's leaders, enacted licensing laws that required as a minimum for medical practice a diploma from a recognized medical school. The real impetus for excellence came shortly after the first World War with the Flexner report, resulting in a much needed overhaul of the educational system. At about the same time, as the practice of medicine became increasingly complex, medical specialty boards began certifying competence in specialized areas; this gained impetus with the vast leaps in technology resulting from research in World War II.

The practice of medicine over the next two or possibly nearly three decades was satisfying, it was fun, and it brought to the American public, as well as to the many from foreign lands who came to seek it, the best medical care available anywhere in the world. The American Medical Association developed a code of ethics that was uniformly adhered to, and on occasions when it was not, there were ethics committees and grievance committees in the local medical societies that dealt with the situation. Though the medical profession was sometimes accused of sheltering its own, sometimes with justification, there were teeth, the ultimate being expulsion, a measure that could bring a medical practice to a screeching halt, as admission privileges at hospitals and even membership on hospital staffs were dependent upon membership in the local society.

The Nashville Academy of Medicine—like, I'm sure, every other medical society—is continually getting calls and letters from patients with a cry of "Can't you *do* something?" or colleagues, particularly the young and inexperienced, ask, "Why don't *you do* something?" about whatever. We don't "do something" because we *can't do* anything. Everything we could once do is in restraint of trade or it interferes with somebody's livelihood (even if it's shady). Try to limit a colleague's activities and you could wind up paying him treble damages. The cry of the public, and the warning from certain regulating agencies for medicine to clean up its act, is nullified and frustrated by other agencies, particularly that watchdog of tradesman, the FTC, who defy us to clean up our act.

We have a lot of problems these days, and this one sometimes pales in comparison, unless one happens to be a member of a grievance committee or the board of trustees of a local society, where the flack sometimes gets heavy. I hate to say it, but some of our colleagues are operating on the ragged edge of respectability—not many of them, but some—and they are charging a lot for it, too. It is quite a comedown from that "golden age" of medicine. We can now, to be sure, do a lot more *for* our patients, but the way has been opened for us to do it *to* them, too.

What's more to the point, the snake oil salesmen are moving into position. Try to keep *them* out, and see where it gets you. The AMA has done away with its department on quackery and questionable practices. It isn't because such don't abound, either.

The bottle is still there; only the picture of the face on it has changed.

J.B.T.

Elementary, My Dear Watson

Perhaps the most persuasive mark of the theater "heavy" is the curled lip. The English seem to do this better than most anyone else (except Charles Boyer); some of my favorites of that genre are (or perhaps better, were) Basil Rathbone (ol' Sir Guy of Gisborne himself), Charles Laughton (who *was* Captain Bligh, destroying with his art the name of an apparently fine officer), Vincent Price, and Charles Sanders. All of them, of course, as superb actors, could and did portray other sorts of characters, but only if a sneer was appropriate.

In addition to being the dastardly Sir Guy, Basil Rathbone *was* Sherlock Holmes for my generation, and a better characterization than his of the great detective is hard to imagine. Now that the goodie oldies are shown regularly on cable channels, I suspect Mr. Rathbone is S. H. to the younger generations, too. That he is a piece of fiction is apt to escape us.

That Sherlock Holmes is a piece of fiction has sometimes escaped a loyal following of his for just a century come November, the date the first Sherlock Holmes tale hit the streets. It was *A Study in Scarlet,* a novel that appeared in *Beeton's Christmas Annual* after it had been rejected by two other publishers, much, I am sure, to their everlasting chagrin, not to mention impoverishment. Mr. Holmes' literary master, Sir (later) Arthur Conan Doyle, M.D., was paid the magnificent sum of £25 for his efforts; he received no royalties. Later on, he did better for himself as Baker Street became an international institution (though there was in fact no 221 Baker Street until a street renumbering in 1930).

It is in *A Study in Scarlet* that Holmes meets Dr. Watson, who becomes his biographer and companion—not so much his straight man as Nigel Bruce makes him on film. Though later studies of Holmes have never equaled that of Basil Rathbone, they have made Dr. Watson less bumbling, an improvement, I think, and truer to Dr. Doyle's account; Watson, a former army surgeon, retired after receiving wounds in India, may have been gullible, but never bumbling.

At the age of 22, Arthur Conan Doyle received his M.D. degree from the Royal Infirmary in Edinburgh, his home town, and after a year as a ship's surgeon on a whaler set up practice with a fellow medical student in Plymouth. His practice, soon solo, never flourished, and he filled in the time between patients with his writing. He abandoned his practice altogether in 1890, and for his efforts was knighted in 1902. He wrote other things than Sherlock Holmes stories, of which there were four long ones and 56 short ones, and some of those others were well received. He tired of Holmes on occasion, and both retired him and tried to do him in, but had to bring him back out of public demand. He has even been accused of being jealous of Holmes. His last story was published in 1927, three years before his death.

Sir Arthur was something of a detective himself, having solved two rather complex cases that resulted in the acquittal of the suspect in each. There is controversy over whether he himself served as his model for either Holmes or Watson. The best estimate is that the model for Holmes, insofar as his deductive abilities are concerned at least, was Dr. Joseph Bell, an Edinburgh surgeon and one of Doyle's instructors. The Sherlock Holmes Society of London, which now has over 600 members and publishes the biannual *Sherlock Holmes Journal,* fosters the notion that Holmes actually existed and that Watson documented his actual cases, often to the discomfiture of Scotland Yard, whose dicks often do appear bumbling in the stories. Doyle is to be looked upon, according to them, as simply the literary agent that arranged for the publication of Watson's memoirs. Perhaps he did well to be jealous of Holmes.

All of which shows how history gets changed. Give 'em another hundred years or so, and Holmes will survive triumphantly; Sir Arthur will have been only his hack. *Sic transit gloria mundi.* The servant is not greater than his master—except sometimes.

J.B.T.

Roadside—Er—*Flowers?*

We are the roadside flowers
Straying from garden grounds,
Lovers of idle hours,
Breakers of ordered bounds.

—Bliss Carmen
Roadside Flowers

A few things still linger, sometimes vividly, in memories of our annual treck from Chattanooga to Daytona Beach when I was young. Colorful chenille bedspreads bedecking clotheslines, making an artist's palette of the sometimes dreary countryside from Dalton to Cartersville. Roadside peaches in South Georgia, and the Perry Hotel (still a haven for the next generation as the "New Perry Hotel and Motel"). Taking forever to get through Atlanta (after more than a half century, and with interstates, it still does). Red clay banks along North Georgia roads, often covered with Kudzu; pecan and peach orchards from Perry to Macon, and sandy fields of jack pine around Waycross, which through North Florida had an undergrowth of palmetto. And Burma Shave.

There was not much in the countryside to entertain small boys. On grounds that it was bad for the eyes (I have yet to figure that one out), reading in the moving automobile was forbidden. My brother and I soon tired of "bull"—the game in which you count cows and have to start over again if you see a white horse—and the scenery, which through Georgia mostly isn't much, in any case. So Burma Shave's little jingles, strung out on five small red signs over maybe a mile, were a welcome and greatly anticipated respite.

Since the scenery along US 41 was virtually unspoilable, and the signs small and unobtrusive—not to mention entertaining—I never heard them vilified; they were finally interdicted as a safety hazard with the onslaught of heavy, high speed traffic, much of it 18 wheelers. I don't remember much about roadside signs generally in those days, except that there were some—probably lots. World War II intervened, and afterwards, when we were traveling the same route with children of our own, there definitely were lots. And not unobtrusive, either.

Tennessee is one of our nation's most scenic states. Grand vistas can unfold before the traveler from the Tennessee River in the west to the Great Smokies and the Blue Ridge Mountains in the east. (The Mississippi Delta has the same scenic properties as South Georgia—not much.) Unfortunately, to have the grand vista unfold one has to abandon the highway and walk at least 50 feet away from it. If one stays on the highway, what is likely to unfold are instructions for getting to the Rise'n Shine Biscuitry or the Stop 'n Slumber Nitery at the next exit, or some such. To be sure, that is not necessarily all bad. The kid-weary traveler can point to the sign that announces, "Twelve Miles," in answer to the strident cries of, "When do we stop?" Small comfort to everybody else. The only others to profit from the experience are the outdoor advertising companies.

Outdoor advertising companies are obviously more potent in Tennessee, at least before its Legislatures, than its millions of citizens, who would rather look at trees than billboards. The outdoor advertisers took to chopping down trees to put up their sightblinders, and irate sightseers took to chopping down billboards to restore the sights. It was a standoff that cost the advertisers a lot of money and the citizens a lot of trees. A compromise was struck by Governor Alexander, which didn't work. We are back at least to losing trees. I don't know about the billboards, since I don't worry much about them. Their owners can obviously take care of themselves—at our expense.

I was struck by this while touring the Northwest and British Columbia last summer, where such desecration is forbidden by law. Until that happens here, Tennessee roadside flowers will continue to be weeds of wood and metal that blossom willy-nilly, at the pleasure of the Legislature's pampered few, to clutter the highways, and even sometimes the byways.

It does keep the driver's eyes on the road. The rest can catch up on their beauty sleep. And the kids? Try "bull."

J.B.T.

our mail box

Gate Keepers

To the Editor:

Your recent editorial in the December 1986 issue of the TMA *Journal,* along with Dr. Eason's article, which complimented each other very nicely, prompted me to make and/or elaborate on a few points regarding the HMO situation in Nashville.

The "gate keeper" arrangement as noted by Dr. Eason is a very important part of the control of the cost of medical care, although it is important that the gate keeper's decisions be based on medical judgment and not financial concerns. We, the medical profession, need to become hard nosed with respect to overutilization, but at the same time we must be very careful that we do not diminish the quality of patient care by trying to save money, particularly for the insurance companies, which HMOs primarily are. The question then becomes who will profit and how, with respect to the financing of the HMO.

The physician-owned and controlled HMO is the ideal HMO solution, if there is any such thing. This entity has the responsibility of controlling the utilization of medical resources; it is responsible for seeing that the patient can see the proper physician, whether it be primary care or specialist, the proper number of times, and for the proper reasons. It also has the responsibility of having a major input into hospital and pharmacy utilization.

This entity does not, therefore, have to make money, though it will if it is successfully operated as a business. This entity sees to it that the physician is paid his fee-for-service, though fees must, of course, be reviewed by reasonable people, who do not allow overcharge for either office visits or procedures. This part of the problem can be controlled by controlling who belongs to the HMO, which means that the medical director has to be somewhat autocratic, but at the same time, exercise his autocracy with some wisdom, common sense, and flexibility. He cannot always be right, and therefore must be willing to listen to his peers.

Whether primary care or specialist, the physician has an equal responsibility with respect to cooperation and utilization. Whether he is an investor or merely has signed on as a provider of care, he needs not only to be interested in having patients provided to him, but to be very conscious that the financial entity needs to be able to pay its business costs. If the physician is not cooperative, does not do outpatient workups, does not carefully control the utilization of the hospital, then financial problems will arise. Simple things, such as hospital admission on the day of a surgical procedure when possible, are certainly desirable. The discharge of a patient in the afternoon, as opposed to the following morning, is important. Many physicians do not understand that the midnight census is most often used by the insurance entity as the time that another hospital day starts.

The capitation programs that Dr. Thomison mentioned seem to me to be truly malignant. There is no way that this country, which has what I believe to be the very highest quality of medical care, can continue to supply such care under a capitation program. The quality can survive if we do away with many of the "frills," some of which can easily be avoided. We physicians have been a willing party to overutilization through the years, when it was so simple to send the bill to the insurance company, who paid it in full. This type of arrangement is no longer possible, though I do not believe that rationing of health care need be the trap that we fall into next.

With respect to capitation, I would like to point out that that system allows the insurance company to put its entire risk upon the physician. The insurance company can know in advance exactly how much it can and will make, without taking any of the risk. It, after all, is in this business to make a profit. If it cannot make a profit, the companies will do as they did here in Tennessee a few years back regarding malpractice insurance. They will simply quit serving this medical community. It therefore means that an amount paid to me per month to care for a given number of patients, with the strings they attach, can do nothing but encourage me to undertreat the patient.

In the first place, the withhold, which the physician-owned and controlled IPA uses to increase capitalization, is used in an entirely different manner by the capitation arrangement. It is used to pay the insurance company for those dollars that you spend on referral to specialists and for hospitalization. Currently, the specialist will, for the most part, receive his fee, although this will have to be negotiated with the insurance company. Actuarially, the insurance company can determine how many patients will need to be hospitalized during a given period of time. They will control this by preadmission certification and by charging the primary care physician part of the hospitalization cost if he allows too many patients to be hospitalized, regardless of the legitimacy of that hospitalization.

One insurance entity here in Nashville, namely Equicor, has so far been unsuccessful in obtaining enough physicians to have a capitation program for sale, and has therefore tried to capitate us by the back door. They have simply taken their PPO arrangement and added a withhold, which withhold will be used to pay any overage in a specialist pool and/or the hospital pool, at the end of the year. They again have all the cards, call all of the shots, and have assured their profit on the front end.

I am as concerned as anyone about the necessity for controlling the cost of medical care. I do not believe that we the physicians can do it alone. The federal government is attempting to control its costs by reneging on political promises and trying to lay the blame on the physician for the increased cost to the elderly patient of his Medicare program. The insurance entities are attempting to do a very similar thing. Somewhere, the public also needs to realize that the cost of medicine is determined by several things, including their utilization of it. They demand care for themselves without understanding that they need to be also concerned about their neighbor. The cost of care is also controlled by such things as the population growth, the cost of "running a hospital," and new technology, as pointed out in a recent special communication in the *JAMA* by Dr. Harry Schwartz of Tufts University School of Medicine. He points out that even after all unnecessary days have been eliminated, the underlying rate of increase will reemmerge unless "limitations are placed on technological innovations, or beneficial services are rationed."

Having been involved from its inception here in Nashville with a physician-owned IPA HMO, I have become increasingly aware that we the profession have the best opportunity we have had in a long time to do our share in controlling the cost of medical care. This will, however, necessitate a great deal of thoughtful and willing cooperation within the profession. It will necessitate things such as preadmission certification, utilization review, and a willingness on the part of the HMO not to renew contracts with "bad actors," whether they abuse the system in terms of overutilization or overcharging. If the insurance company can make a guaranteed profit by having us in servitude, then certainly we can do an even better job as free men.

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To the Editor:

Over the past year I have read with great interest your pointed editorials, especially when you discuss issues pertinent to the "health care revolution." Your most recent article in the December 1986 issue of our *Journal* points out succinctly the potential grave pitfall for all physicians involved in capitated medical care. Health insurance, as any insurance, is purchased by the buyer to protect himself from financial catastrophe. I agree with you that it is unfair and unwise to make physicians their patients' advocates and insurers at the same time through capitation of medical care. Pertinent to this also is the recent TMA position regarding establishing an IPA HMO, which I also feel is a grave error in judgment. There is no doubt that there is a great need for physicians to monitor their colleagues and eliminate waste in medical care through appropriate use of hospital or outpatient facilities determined by their patients' needs. Clear examples of substantial savings in health care dollars doing just that are available almost anywhere you look. The risk-sharing features of HMOs, whether they be true capitation of a primary care gate keeper or capitation and risk of a group of physicians in an IPA, is a risky business at best, not only for the physicians involved but potentially for their patients also. Although I do participate in HMOs, I do so in a careful and selective manner with the jitters in my stomach, to perhaps learn from the experience and also perhaps altruistically not to deny some of my patients continued follow-up (albeit not by me necessarily) in a changing health care system.

Keep the comments coming! Although our dog in this fight seems to be losing to the more aggressive corporate and government beasts, the outcome has not yet been determined, and perhaps, for the betterment of the health care of our patients, we can still make appropriate changes in health care delivery without conflicts of interests.

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To the Editor:

This is in response to the editorial of Dr. John B. Thomison and the Special Communication from Dr. Hamel B. Eason, both of which appeared in your December 1986 edition of the *Journal.* They were informative and challenging. Taken together they demonstrate the range of understanding and misunderstanding extant in the medical profession about the changes that currently plague us.

At one extreme is the constructive evaluation of reality represented in Dr. Eason's remarks, wherein he correctly identifies that change is driven by legitimate concerns within society about the cost of medical care. As a consequence those who purchase health care services have sought to alter the financial incentives and the control procedures that impact physicians since physicians allocate the resources in health care. These are normal and predictable responses that would be expected in other endeavors in our society. Some of the initiatives being urged upon us are wise and workable; others are not, as Dr. Eason indicates. However, none of them are unusual nor necessarily good or evil.

At the other extreme of reaction seen in medicine today is the diatribe of Dr. Thomison's editorial. We in medicine look ingenuous, if not desperate, when we sweepingly attack the morality of "business men." To label HMOs as "that anathema of good medical care" is equally desperate in tone, and disregards the fact that tens of thousands of fine physicians in excellent health care organizations are practicing in HMOs. They are doing it without prostitution of their professional standards. Evidence in the peer review literature indicates that their patients' health remains equal to that of others who receive care in non-HMO settings. We are on very thin ice when we call capitation for the payment of services "a license to steal." This is especially true since the alternative financing system for payment so frequently espoused by our profession is that of fee-for-service. The author of this editorial has not been to many cocktail parties if he has not been subjected to the same complaint or joke about "the doctor's fee" usually delivered only half in jest.

At a recent meeting I attended there were present physicians, health care consumers, medical administrators and persons from several constituent organizations concerned with health care policy, including both quality and cost issues. As part of one panel I heard a physician leader complaining about the changes occurring in medicine with frequent prediction about the unfavorable results that he foresaw relative to quality of care. As he warmed to his subject he finally predicted that if the government and business didn't back off from their efforts to change the way medicine is practiced, "Doctors are going to take to the streets!" It was then that I witnessed a frightening and sobering response. In reply a lady from a senior citizens organization replied, "Well Doctor, I predict you won't find anyone out there to support you but other doctors."

At the December 1986 interim meeting of the American Medical Association, Dr. James Sammons identified the fact that there remains a significant reservoir of distrust of the motives of physicians within American society. This is mentioned here to underscore the critical point that the shape and form of the health care system of the future will not be a product solely or even mainly of the medical profession's preferences or biases. Health care is a business as Dr. Thomison posits. The record shows that consumers who have been participants in an alternative delivery system (HMO, PPO, EPO) adapt to that style of care and tend to stay with it—or some other form of managed health care—when they subsequently change their health insurance arrangements. Although most physicians abhor the very idea, health care is becoming market driven. The "golden age"' of a seller's market, enjoyed by doctors but a few years ago, is not going to return. The growing supply of physicians will assure that. The sad fact for us in medicine is consumers and those who purchase health care will define the health care system of tomorrow.

All of this is a staggering challenge to our profession. The task so clearly before us is to define a constructive agenda that will allow the profession to move through this difficult time and maintain the essential values of the profession in a time of change. Attacking the "morals" of the business community and other such polemics is not that constructive agenda.

The problem in health care today is not the quality of the health care system. The problem is how to pay for the system that has been built over the past 25 years. That problem has not been successfully addressed in spite of HMOs, PPOs, or DRGs. The rate of increase in the cost of health care remains two or three times that of the remainder of the economy. Buyers of health care (individuals, the government, industry, labor unions) continue their search for a control mechanism. The simple process of compounding rate of increase must make the necessity for control evident to any thinking person. The insurance companies, in the absence of any others prepared to undertake the task, are the agents of change who operate now to effect the will of those who pay the bill.

Insurance companies have a difficult task before them. That task is to halve (at least) the rate of growth in the cost of health care while maintaining the desired level of quality within the system. The tools insurance companies are using in this endeavor are financial incentives and medical case management. These companies are not wedded to any of the individual initiatives involved such as capitation, risk pools, preadmission review, etc. They are willing and ready to pay physicians, hospitals or any combination thereof for quality and cost-effective health care for their constituents, who are health care consumers and buyers. Insurance companies would be pleased to delegate the role of change agent to some other demonstrably effective agent. Those responsibilities are costly and controversial. It would be very comfortable for them to return to an arm's length relationship such as existed when we sent the bill and they had only to pay it in full to please everyone—the patient, the provider, and the buyer.

That is the challenge and the opportunity for physicians today. We in medicine need to get ourselves organized and go to work at the task of fashioning a quality, cost-effective health care system in response to the needs of the marketplace. It is essential that we stop grieving about the "good old days" and recognize that it is perfectly possible to build a quality, cost-effective system using different reimbursement and control procedures. In that process we can fashion a system that will retain for physicians clinical autonomy, plus the right of final judgment about quality of care.

Dr. Eason referenced physician-friendly and physician-controlled IPAs (PF/PC-IPA) as the workable model for the alternative delivery and financing system. He is very much on target. However, we must realize that the PF/PC-IPA has the same mission as any other alternative system, i.e., halve the rate of growth of the cost of health care while retaining the quality demanded in the market. That can be done. Alone or more often in league with insurance companies, it's being done by doctor-controlled organizations today. It requires change—inescapable change demanded by society. Positive initiatives to that end by us in medicine would be enthusiastically welcomed by all involved in this complex problem—consumers, buyers, and insurer alike.

David J. Ottensmeyer, M.D.
Chief Medical Officer
Equicor
P.O. Box 1115
Nashville, TN 37202

Reply

I have stated my position. Dr. Ottensmeyer has provided me with no reason to alter it.

John B. Thomison, M.D.
Editor

in memoriam

J. B. Hibbitts, Jr., age 92. Died January 10, 1987. Graduate of Johns Hopkins University Medical School. Member of Nashville Academy of Medicine.

Thomas Wheeler Johnson, age 62. Died January 3, 1987. Graduate of University of Tennessee College of Medicine. Member of Northwest Tennessee Academy of Medicine.

Gerald I. Plitman, age 61. Died December 20, 1986. Graduate of Vanderbilt University School of Medicine. Member of Memphis-Shelby County Medical Society.

William Cecil Stanbery, age 90. Died January 9, 1987. Graduate of University of Tennessee College of Medicine. Member of Bradley County Medical Society.

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

BRADLEY COUNTY MEDICAL SOCIETY
Michael Alan Daubner, M.D., Cleveland

CONSOLIDATED MEDICAL ASSEMBLY OF WEST TENNESSEE
Tim Linder, M.D., Lexington

FRANKLIN COUNTY MEDICAL SOCIETY
Diane L. Petrilla, M.D., Sewanee

KNOXVILLE ACADEMY OF MEDICINE
Samuel I. Brown, M.D., Knoxville
James L. Burns, Jr., M.D., Knoxville
Andrea J. Eberle, M.D., Knoxville
John E. B. Harrison, M.D., Knoxville
Thomas T. Kubota, M.D., Knoxville
Gloria L. Lewis, M.D., Knoxville
Kuang-Tzu Davis Lin, M.D., Knoxville
Chris John Martin, M.D., Knoxville
Christopher A. Miller, M.D., Knoxville
David B. Reath, M.D., Knoxville
Reynald T. Simmons, M.D., Knoxville

MONTGOMERY COUNTY MEDICAL SOCIETY
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NASHVILLE ACADEMY OF MEDICINE
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Mary Anne Blake, M.D., Nashville
Mary Jane Brown, M.D., Brentwood
John Charles Dalton, M.D., Nashville
Ben Weldon Davis, M.D., Nashville
Edwin Boyette Emerson, M.D., Nashville
Jack Fisher, M.D., Nashville
William Harrison Frist, M.D., Nashville
Bruce Randolph Goodman, M.D., Nashville
Robert William Herring, Jr., M.D., Nashville
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Jeffrey Lawrence Hymes, M.D., Nashville
James Duncan Ladd, M.D., Nashville
Carolyn M. Parrish, M.D., Nashville
William Edward Watson, M.D., Nashville

(Student)

Susan L. Wouters, Nashville

ROANE-ANDERSON COUNTY MEDICAL SOCIETY
Daniel E. Conrad, M.D., Oak Ridge
Floyd E. Thurston, M.D., Oak Ridge

RUTHERFORD COUNTY/STONES RIVER ACADEMY OF MEDICINE
Paul David LeDoux, M.D., Murfreesboro

SULLIVAN COUNTY MEDICAL SOCIETY
Susan M. Bland, M.D., Kingsport
Jim C. Brasfield, M.D., Bristol
James Richard Cavett, III, M.D., Bristol
Jeffery Dale Sargent, M.D., Bristol
Harold A. Sims, M.D., Bristol
William David Smith, M.D., Bristol
Barry E. Wright, M.D., Bristol

SUMNER COUNTY MEDICAL SOCIETY
Anthony V. Dallas, Jr., M.D., Hendersonville

WASHINGTON-UNICOI-JOHNSON COUNTY MEDICAL ASSOCIATION
Gregory Gene Ames, M.D., Jonesboro
Frank P. Johnson, Jr., M.D., Johnson City

WHITE COUNTY MEDICAL SOCIETY
William Allen, M.D., Sparta

WILLIAMSON COUNTY MEDICAL SOCIETY
Henry P. Russell, M.D., Franklin

personal news

Paul C. Frederick, M.D., Jackson, has been certified as a Diplomate of the American Board of Obstetrics and Gynecology.

Jack M. Roe, M.D., Livingston, has received the Livingston Civitan Club's annual Citizenship Award, which is presented to a person the club feels has been of outstanding service to the community.

Robert B. Wilson, M.D., Huntingdon, has received Carroll County's highest honor, the coveted Silver Medallion Award, recognizing his more than 50 years of service and dedication.

TMA Members Receive AMA Physician's Recognition Award

Twelve TMA members qualified for the AMA Physician's Recognition Award during December 1986.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Merton M. Beckwith, M.D., Nashville
Ralph L. Brickell, Jr., M.D., Tullahoma
John E. Chapman, M.D., Nashville
James D. Crutchfield, M.D., LaFollette
William E. Feist, M.D., Chattanooga
David N. Jones, M.D., Nashville
Merrill D. Moore, M.D., Knoxville
James R. Moyers, M.D., Nashville
Raphael F. Smith, M.D., Nashville
Mary E. Thompson Stroud, M.D., Chattanooga
Ronald L. Terhune, M.D., Memphis
Melborne A. Williams, M.D., Nashville

announcements

CALENDAR OF MEETINGS

NATIONAL

April 11-13	American Society for Laser Medicine and Surgery—Hyatt Regency Embarcadero, San Francisco
April 19-24	American Fracture Association—Chicago
April 23-27	American Medical Society on Alcoholism and Other Drug Dependencies—Stouffer's, Cleveland
April 25-26	American Laryngological Association—Fairmont, Denver
April 25-May 1	American Occupational Medical Association—Wyndham Franklin Plaza, Philadelphia
April 27-28	American Broncho-Esophagological Association—Denver
April 27-30	American College of Obstetricians and Gynecologists—Las Vegas
April 27-30	Southwestern Surgical Congress—Coronado, Calif.
April 27-May 1	Ambulatory Pediatric Association—Anaheim, Calif.
April 27-May 1	Society for Pediatric Research—Anaheim, Calif.
April 28-30	American Laryngological, Rhinological, and Otological Society—Denver
April 28-30	American Society for Head and Neck Surgery—Fairmont, Denver
April 29-May 2	American Burn Association—Washington, D.C.
April 30-May 3	American Association for the History of Medicine—Philadelphia
April 30-May 3	Christian Medical Society—International Hotel, New Orleans
May 1-4	American Federation for Clinical Research—Town & Country, San Diego
May 1-4	American Society of Clinical Investigation—Town & Country, San Diego
May 1-4	Association of American Physicians—Town & Country, San Diego
May 3-7	American Association of Neurological Surgeons—Dallas
May 3-7	Uniformed Services Academy of Family Physicians—Orlando, Fla.
May 4-8	Association for Research in Vision and Ophthalmology—Hyatt, Sarasota, Fla.
May 5-7	American College of Legal Medicine—Long Beach, Calif.
May 6-10	American Psychoanalytic Association—Hyatt Regency, Chicago
May 7-10	American Academy of Psychoanalysis—Chicago
May 9-13	American Society for Gastrointestinal Endoscopy—Hyatt Regency, Chicago
May 9-15	American Gastroenterological Association—Hyatt Regency, Chicago
May 9-15	American Society of Neuroradiology—New York
May 10-13	American Thoracic Society—Marriott/Sheraton, New Orleans
May 10-13	Society for Cardiovascular Anesthesiology—Marriott, Palm Desert, Calif.
May 10-14	Aerospace Medical Association—Las Vegas
May 11-13	Society for Surgery of the Alimentary Tract—Chicago
May 12-15	Society for Cardiac Angiography—Disneyland Contemporary Hotel, Orlando, Fla.
May 14-17	American Geriatrics Society—Hyatt Regency, New Orleans
May 14-17	Association of Clinical Scientists—Charleston Place Hotel, Charleston, S.C.
May 14-17	Virginia Society of Ophthalmology, annual meeting—Sheraton Tysons Corner, Northern Virginia
May 17-20	American Ophthalmological Society—La Costa, Carisbad, San Diego
May 17-21	American Urological Association—Hilton, Anaheim, Calif.
May 19	American Medical Student Association—New Orleans
May 19-21	American Society for Artificial Internal Organs—Marriott Marquis, New York
May 23-27	American Fracture Association—Hyatt Regency, Chicago
May 24-28	American Association on Mental Deficiency—Hilton, Los Angeles
May 25-30	American Association of Suicidology—San Francisco
May 26-27	American Otological Society—Fairmont, Denver
May 26-30	American College Health Association—Chicago
May 27-30	American College of Sports Medicine—Las Vegas

TMA continuing medical education opportunities

The continuing medical education accreditation program of the TMA has full approval by the Accreditation Council for Continuing Medical Education. An accredited institution or organization may designate for Category 1 credit toward the AMA Physician's Recognition Award those CME activities that meet appropriate guidelines. If you wish information as to how your hospital may receive accreditation, write: Director of Continuing Medical Education, Tennessee Medical Association, 112 Louise Ave., Nashville, TN 37203.

IMPORTANT NOTICE

Published in this section are all educational opportunities which come to our attention which might be of interest to our membership. As some of these are very long, full year schedules, and others are detailed descriptions of courses, in order to conserve space, most of them will be published in only one issue of the Journal.

IN TENNESSEE

VANDERBILT UNIVERSITY

Clinical Training Program

Opportunities for advanced clinical education for physicians in various subspecialties have been developed by the School of Medicine and the Division of Continuing Education of Vanderbilt University. The practicing physician, with the guidance of the faculty from the participating department, can plan an individualized program of one to four weeks to meet recognized needs and interests. The experience may include contact with patients; interaction with clinical and full-time faculty, housestaff, and fellows; participation in clinical rounds and diagnostic procedures. There is access to the Medical Center Library with excellent resources. Experience in more than one discipline may be included if time permits. *Eligibility:* Licensed physicians in active practice are eligible to apply. *Credit:* AMA Physician's Recognition Award (Category 1) and/or AAFP Continuing Education Accreditation. *Application:* For information and an application contact Marilyn D'Asaro, Division of Continuing Medical Education, Vanderbilt University School of Medicine, CCC-5326 MCN, Nashville, TN 37232, Tel. (615) 322-4030.

Continuing Education Schedule

April 24-25	Annual Barney Brooks Lecture and the H. William Scott Society
May 22-23	11th Annual Sonography Symposium
May 26-30	Family Medicine Review
May 31-June 7	British American Cardiology at the Hammersmith—London, England
June 3-6	Advances in Medical Imaging—Kaiwah Island, S.C.
June 18-19	Parkinson Disease
July 21-25	10th Annual Contemporary Clinical Neurology—Hilton Head Island, S.C.

For information contact Division of Continuing Medical Education, Vanderbilt University School of Medicine, CCC-5326 MCN, Nashville, TN 37232, Tel. (615) 322-4030.

MEHARRY MEDICAL COLLEGE

Extended Continuing Education Program

Arrangements have been made with the following services and departments in the medical school to allow practicing physicians to participate in that service's activities for a period of one day to one week. This program provides an opportunity for physicians to study in depth for a specified period. The schedule of activities is individualized in response to the physician's request by the participating department. The experience includes conferences, ward rounds, audiovisual materials and contact with patients, residents, and faculty.

Fee: $75 per day or $275 per five day week of educational experience including lunch and parking at Meharry Medical College. *Credit:* AMA Category 1 of the Physician's Recognition Award, AAFP, American Academy of Family Physicians and Continuing Education Units from Meharry Medical College. *Application:* For information contact Dr. Henry A. Moses, Ph.D., Director of Continuing Education, Meharry Medical College, 1005 D.B. Todd Boulevard, Nashville, TN 37208, Tel. (615) 327-6235.

UNIVERSITY OF TENNESSEE

Continuing Education Schedule

Memphis

April 1-4	Critical Care Medicine—Hot Springs, Ark.
April 9	College of Medicine Alumni Reception—Knoxville
April 23-25	Pediatric Critical Care
May 6-7	M. A. Reeves Distinguished Visiting Professorship in Nutrition
June 12-14	College of Medicine 30 Year Reunion

Knoxville

April 31-May 2	Family Practice Update—Gatlinburg

Chattanooga

April 24	Resident Research Day
May 14-15	Current Concepts in Cancer
June 24-27	Family Medicine Review
Aug. 6-7	Infectious Diseases—Williamsburg, Va.
Aug. 12-15	Menopause: Consequences and Treatment Strategies—Napa Valley, Cal.
Oct. 19-24	Emergency Medicine Review

For information contact Mrs. Jean Taylor Bryan, Office of Continuing Medical Education, University of Tennessee College of Medicine, 800 Madison Ave., Memphis, TN 38163, Tel. (901) 528-5547.

EAST TENNESSEE STATE UNIVERSITY

April 2	School Health
April 9-10	16th Annual School Health Education
April 23-25	Cardiology
May 30	Tough Psychiatry Problems in Medical Practice—Gatlinburg

For information contact Ramona Miller, Ph.D., Program Coordinator, Office of CME, Quillen-Dishner College of Medicine, Box 19660A, Johnson City, TN 37614, Tel. (615) 929-6204.

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Perinatal Management Strategies For Fetal Gastroschisis

MICHAEL S. CARDWELL, M.D.

Gastroschisis is an uncommon fetal anomaly with an incidence of 1 per 12,500 births.[1] Prenatal diagnosis via obstetrical ultrasonography is possible.[2] Elevated maternal serum alpha-fetoprotein (MSAFP) may also suggest the presence of fetal gastroschisis.[3]

The differential diagnosis of gastroschisis includes omphalocele, amniotic band syndrome, and bladder (cloacal) extrophy.[4] Gastroschisis is usually an isolated defect and is readily remediable by operative therapy provided that a timely diagnosis is made and appropriate obstetrical-neonatal intervention is carried out. The following case emphasizes many of the perinatal management strategies that will optimize the outcome of a pregnancy complicated by fetal gastroschisis.

Case Report

An 18-year-old primigravida whose menstrual dates were uncertain was hospitalized for pyelonephritis on Jan. 21, 1986; obstetrical ultrasonography suggested a gestational age of 18 weeks. No gross fetal anomalies were noted. An MSAFP, ordered by the obstetrical consultant as part of his routine prenatal evaluation, was 268 ng/ml, 2.5 times greater than multiples of the median (MoM) for the reference laboratory. The patient was referred to the Maternal-Fetal Medicine Department for further evaluation.

A repeat obstetrical ultrasound examination with real time scanning revealed a fetus of approximately 17 to 18 weeks with freely floating intestines in the amniotic fluid cavity. No covering membrane was present. No other gross anomalies were identified. The patient was counseled regarding the findings, and genetic amniocentesis was recommended to verify chromosomal normalcy of the fetus. Amniocentesis under ultrasound guidance, with subsequent karyotyping, revealed a normal 46XX female. It was the patient's desire to continue the pregnancy.

Serial ultrasound examinations showed a low growth profile of the fetus, and at approximately 36 weeks of gestation, based on previous ultrasound examinations, the fetus became less reactive on nonstress testing. Estimated fetal weight by ultrasonography was 2,125 gm. An amniocentesis to assess fetal pulmonary maturity showed a phosphatidylglycerol (PG) greater than 2%.

The patient underwent abdominal delivery on June 4, 1986 of a 2,200-gm female infant with Apgar scores at 1 and 5 minutes of 9 and 9 respectively. Gastroschisis was verified on examination, the defect measuring approximately 4 cm in length with only bowel protruding through the defect. No other abnormalities were present, and primary closure of the defect was performed successfully within the first 12 hours of life. The infant did not demonstrate any respiratory difficulty and was discharged in good condition at 5 weeks of age.

Discussion

Abdominal wall defects of the fetus may be serendipitously discovered by obstetrical ultrasonography or may be suggested by the presence of an elevated MSAFP. Like neural tube defects, any disruption of the fetal integument may allow transudation of alpha-fetoprotein from the fetal circulation into the amniotic cavity. Absorption into the maternal circulatory system causes an elevation of the MSAFP.

From the Department of Maternal-Fetal Medicine, Baptist Hospital, Nashville.

Reprint requests to Department of Maternal-Fetal Medicine, Baptist Hospital, 2010 Church St., Suite 223, Nashville, TN 37236 (Dr. Cardwell).

The etiology of fetal gastroschisis is thought to be due to a disruption of the omphalomesenteric artery, which supplies the ventral abdominal wall.[5] Lack of vasculature to the affected region prevents normal embryological closure. Gastroschisis is usually an isolated defect, unlike fetal omphalocele, which may be associated with chromosomal abnormalities, severe cardiovascular anomalies, and specific syndromes such as the Beckwith-Wiedeman syndrome.[6]

Differentiating between gastroschisis and ruptured omphalocele may be difficult with prenatal ultrasonography.[7] An omphalocele usually involves the umbilical cord, whereas gastroschisis is usually located to the right of the umbilical cord. Genetic amniocentesis is recommended in order to detect lethal chromosomal anomalies such as trisomies 13, 15, 18, which may be associated with an omphalocele but not with gastroschisis. Chromosomal analysis is indicated even in the third trimester to prevent unnecessary operative procedures for such lethal aneuploidies. Fetal echocardiography is recommended to detect cardiovascular defects, which may also be associated with fetal omphaloceles.[8]

Intrauterine growth retardation frequently occurs in fetuses with gastroschisis.[9] Transudation of fetal proteins into the amniotic fluid may contribute to nutritional deprivation of the fetus.[10] Serial ultrasound examinations for estimating the fetal weight are helpful in the management of the pregnancy, and may allow a timely delivery prior to a severe growth deficiency.

There is controversy as to the appropriate mode of delivery of the fetus with gastroschisis. Although a recent series by Carpenter and associates[9] of 38 cases of fetal ventral wall defects concluded that vaginal delivery poses no risk to the fetus, other reports[11] suggest that abdominal delivery prevents further trauma to the exposed abdominal contents and prevents contamination from the vaginal flora. In addition, an elective abdominal delivery ensures that the neonatal team, i.e., neonatologists, pediatric surgeons, and others, would be immediately available for care of the newborn. Delivery in a perinatal-neonatal center is recommended, as the mortality and morbidity is much lower for inborn versus outborn, transported neonates.[12]

Summary

In conclusion, prenatal diagnosis of gastroschisis will become more common as MSAFP screening becomes more widely available. Serendipitous diagnosis will continue to be made on obstetrical ultrasound examination performed for unrelated indications. Differentiation between gastroschisis and other fetal abdominal wall defects is necessary in order to appropriately manage the pregnancy. Chromosomal analysis of the fetus is recommended to prevent unnecessary operative delivery. Serial ultrasonography allows assessment of fetal growth and early detection of intrauterine growth retardation. Elective abdominal delivery may prevent further trauma to the fetal gastrointestinal tract, reduce infection, and allow immediate assessment and therapy of the neonate by the complete neonatal team. Delivery in a tertiary center is necessary for optimal outcome.

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Gonadal Failure: A Review

DAVID MURAM, M.D. and THOMAS STOVALL, M.D.

Gonadal failure is a complex subject of importance to the obstetrician-gynecologist as well as to other medical specialists. Gonadal failure encompasses a wide spectrum of causes, phenotypic expressions, and symptomatology, as well as a spectrum of gonadal morphology and function. Gonadal failure comprises between 4% and 10% of all patients with amenorrhea and is responsible for approximately 60% of those with primary amenorrhea.[1] The purpose of this paper is to review the various defects leading to gonadal failure.

Embryology of the Gonad

In order that one can fully comprehend the specific defects in gonadal function, it is necessary to have an understanding of the normal development and function of the gonad. Sex differentiation involves both genetic and hormonal events (Table 1). The human gonads develop as thickenings of the coelomic epithelium over the urogenital ridge, an area composed of germinal epithelium and its underlying mesenchyme. Proliferation of this mesenchyme occurs when migratory primordial germ cells reach the area from the yolk sac.[2] The migratory stimulus remains unknown.

The differentiation of the testes is dependent on the presence of the Y chromosome; it carries the genetic material necessary for the formation of the HY antigen,[3,4] which can be found on the membranes of all 46XY cells and functions to direct the bi-potential gonad to become a testis. When the HY antigen is present and functioning properly, testicular differentiation is induced; in its absence, an ovary is formed.[5]

Even though the development of the bi-potential gonad into a testis or ovary is dependent on the chromosomal makeup of the fetus, it is indistinguishable until six weeks of embryonic life.[6] By the sixth week, cords of epithelial cells mixed with germ cells are seen. The testis becomes distinctive during the seventh week by its complex pattern of sex cords and by its less cellular outer cortical zone, which then forms the tunica albuginea. An ovary can be recognized at this time only because of its failure to show these changes, and it becomes histologically distinguishable at about ten weeks. The sex cords form the seminiferous tubules in the male and the primary ovarian follicles in the female; the mesenchyme forms the Leydig cells in the male and the ovarian stroma in the female.[2]

The presence of two X chromosomes is necessary for complete development and proper function of the ovary. The primary sex cords of the future ovary degenerate, yet remain as recognizable structures until the eighth month. The germ cells increase in number until the 15th week, with little increase in mesenchyme. The primary follicles become recognizable in the fourth month, as they become enclosed with granulosa cells and surrounded by mesenchymal cells which form the theca.[7] The ovary at five months of gestation contains about 7 million germ cells and by birth the number of oocytes has decreased to 2 million. These oocytes remain at the first meiotic prophase until the time of ovulation.[8]

Owing to an intrinsic programming of all genital primordia, the fetal genital tract develops along female lines unless maleness is actively imposed before eight weeks of gestation. During this critical stage of embryonic development, the fetus has two pairs of internal genital ducts, the

TABLE 1

SUMMARY OF EARLY GONADAL DEVELOPMENT

4 weeks—	Primordial germ cells, originating in yolk sac endoderm migrate to the genital riose
5 weeks—	Indifferent gonads are located between the mesonephral and the urogenital riose
6 weeks—	Primary sex cords invade gonads
7 weeks—	If XY, male characteristics appear
12 weeks—	If XX, definite ovary formed, degeneration of primary sex cords

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mullerian ducts and the wollfian ducts. The mullerian ducts are sensitive to mullerian inhibiting factor (MIF), but after eight weeks this inhibiting factor will not prevent the development of the mullerian ducts. Without the MIF, a uterus results regardless of the phenotypic sex of the individual.[9,10]

In order for the 46XY embryo to develop into a normal male, the testis must produce two substances: androgens and MIF. The MIF prevents further development of mullerian structures, which remain rudimentary structures in the male. Testosterone production begins by the eighth week, and in addition to wollfian duct stimulation, it influences descent of the testes from the upper abdomen into the inguinal area by the 12th week.[11,12] The testes reach the scrotum by the 28th week.[13-15] The MIF may influence testicular descent as well, since patients with deficient MIF function commonly have undescended testes.[16] In addition, androgens virilize the external genitalia.

The external genitalia require such androgenic stimulation to develop along male lines. The degree of virilization depends on the amount of androgens and on the timing and duration of exposure (Table 2). In the absence of androgenic stimulation, the external genitalia will develop along female lines. When testes involute following a period of production of MIF and/or androgens, the clinical findings are dependent upon the degree of previous testicular activity and the timing of involution in relation to the critical periods of gonadal development. Thus, when either MIF or testosterone is not produced in sufficient quantity, or when the hormones do not function properly, a female or predominantly female phenotype appears in an individual with a 46XY karyotype.

TABLE 2

TIMING OF TESTICULAR REGRESSION AND ITS EFFECTS ON THE GONADAL TRACT

Time Post-Fertilization Testicular Regression Occurs	Syndrome Produced
Before 43 days	Pure gonadal dysgenesis
43-59 days	Swyer syndrome
60-69 days	Agonadism
70-75 days	Testicular dysgenesis
75-84 days	Testicular regression
90-120 days	Rudimentary testis
After 120 days	Anorchia—normal phenotypic male, no testicular tissue

Putting these events together, if the chromosomes are XY, testes form and if these testes function normally and produce androgens and MIF, the genitalia become male. If there is no Y chromosome, testes do not form, and in the absence of androgens and MIF, the genitalia then become female (Tables 3 and 4). Thus with such a complex chain of events, a variety of abnormalities are possible.

Agonadism

Agonadism affecting the genetic male is a rare syndrome first described by Overzier and Linden.[17] These individuals have amenorrhea, minimal breast development, sparse pubic hair and ambiguous external genitalia, indicating some inutero exposure to androgens. These individuals have small, underdeveloped mullerian structures and no testicular tissue can be found. Removal of the fibrous gonadal ridges is necessary to prevent future neoplasia.[18]

This syndrome can be explained as an insult to the developing testes during the critical period of 60 to 69 days. MIF has been produced but the testis is destroyed before significant amounts of testosterone are produced. Thus, the absence of mullerian duct development and minimal or no virilization. As adults, these patients have elevated gonadotropins, predominantly FSH; cortisol, 17 hydroxyprogesterone, and testosterone are all well in the normal female range.[19] These patients are usually reared as female.

Treatment of these patients can include lengthening the vagina with separation of labial fusion. Estrogen therapy should be employed to feminize these girls, as well as to prevent the development of osteoporosis. The mode of inheritance is unknown, although agonadism has been reported in siblings.[20,21] Agonadism in 46XX individuals resembles the clinical features of 46XX pure gonadal dysgenesis described elsewhere in this review.

Gonadal Dysgenesis

Since Turner first described the Turner syndrome and Ford described the 45XO karyotype many forms of gonadal dysgenesis have been described.[22,23] Gonadal dysgenesis includes a wide spectrum of syndromes including pure gonadal dysgenesis (46XY, 46XX), mixed gonadal dysgenesis (46XY/45X etc.), partial deletions of X [46Xdel(Xq) ring X], structured anomalies of the

TABLE 3

GONADAL FAILURE BASED ON THE HY ANTIGEN

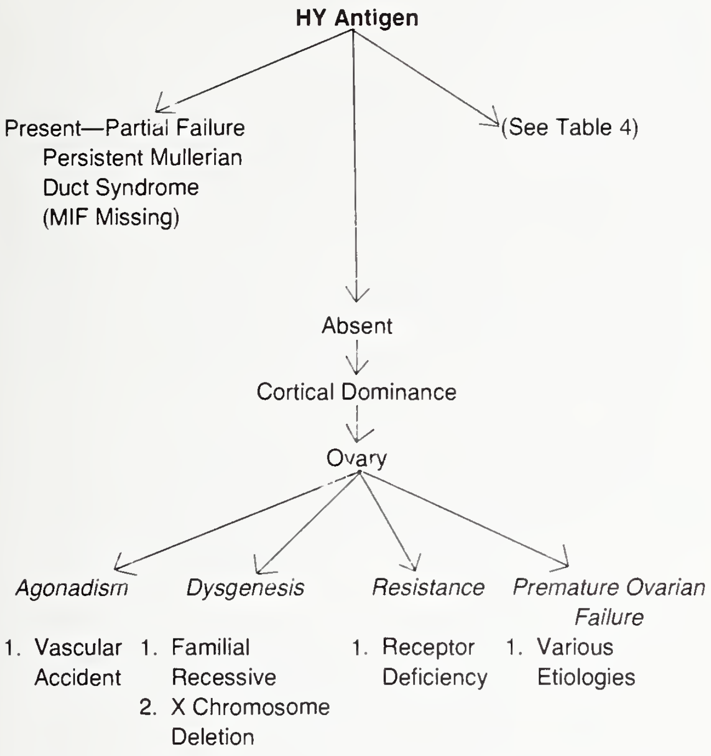
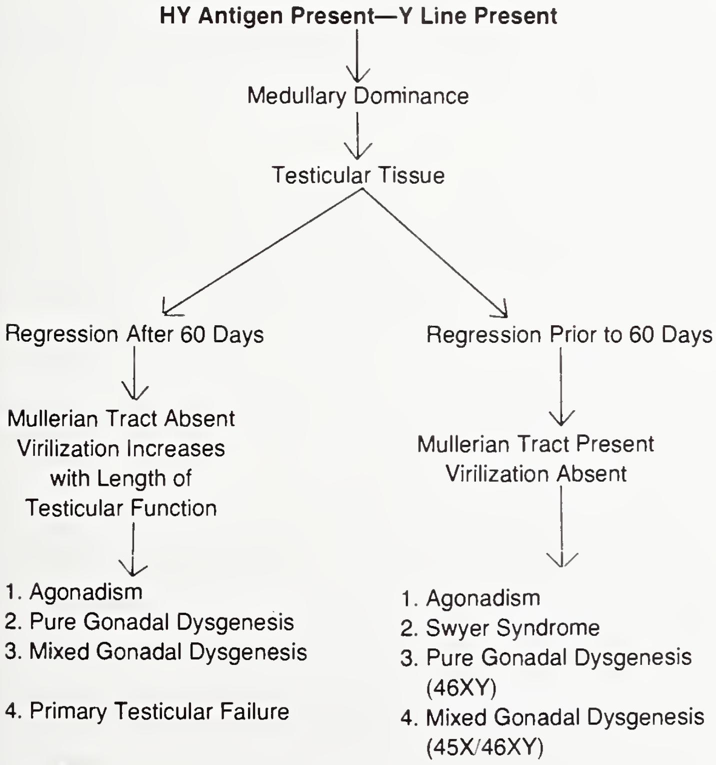


TABLE 4

GONADAL FAILURE: Y LINE PRESENT



X chromosome [46X, iso (Xq)]. Mosaicism is more common than a single cell line. The most common single cell line is 45X followed by 46Xiso(Xq).[24]

The gonads are usually of the streak type and lack follicles and germ cells. However, in up to 30% of patients with 45X karyotype some degree of ovarian function remains throughout puberty, secondary sexual development may occur, and pregnancies have been reported.[25-27] In these individuals ovarian function fails early in life, and the diagnosis is made when premature ovarian failure is diagnosed.

When X chromosome material is missing, these patients usually are of short stature, since height determinants are present on the short arms of both the X and Y chromosomes, and possibly on the long arm of the X. Short stature results no matter what portion of the sex chromosome material is deleted.[28]

Turner syndrome (45X) is the most common form of gonadal dysgenesis and is the most common cause of primary amenorrhea. Patients have varying phenotypes with features that include the most common of all features—short stature—and in addition, coarctation of the aorta, ventral septal defect, brachycephaly, arched palate, cubitus valgus, horseshoe kidney, nail hypoplasia, unilateral renal agenesis, lymphedema, pigmented nevi, pterygium celi, shield chest, short fourth metacarpal.[29] Hashimoto's thyroiditis is associated with isochromosome of the long arm of the X.[30]

Mixed gonadal dysgenesis, the most common form of which is 45XO/46XY, results from a failure of the Y to segregate normally. Thus, in a 46XY zygote this will produce a 45XO/46Y karyotype. The second most common type of mixed gonadal dysgenesis is 45X/46Xiso(Xq). These patients have varying phenotypes which are dependent on the degree of testicular development as well as the amount of androgens produced. Individuals with 45X/46XX karyotype have fewer anomalies than do 45X individuals. Approximately 18% of these patients show some breast development as opposed to only 5% of patients with 45X, and the mean adult heights are greater. The 45X/47XXX karyotype is rare but these individuals are phenotypically similar to 45X/46XX patients. This chromosomal anomaly is the result of an anaphase lag. A deletion of the short arm of the chromosome, 45X/46X del (Xp) often, but not always, results in gonadal dysgenesis and other features of Turner syndrome. Thus the somatic anomalies associated with Turner syndrome are due to a loss of genetic determinant carried on the short arm of the X chromosome.[31]

Approximately 40 cases have been reported with deletion of the long arm of the X chromosome. Menstruation occurs in approximately 20% and breast development in about 25%. The degree of gonadal function cannot be correlated with the amount of Xq material loss. Fifty percent of these patients have normal height.[32]

Familial gonadal dysgenesis has also been described. The condition may be found in phenotypic sisters in whom the karyotype is 46XY or 46XX. The former condition is transmitted by unaffected female carriers in X-linked recessive fashion, whereas the latter is transmitted as an autosomal recessive disorder. However, since patients with 46XY familial gonadal dysgenesis are infertile and male-to-male transmission has not been documented, it is theoretically possible that a clinically indistinguishable disorder exists which is transmitted in autosomal dominant male-limited fashion. In this form of inheritance, the defective dominant gene is situated on an autosome, but its clinical expression is limited to the male, although an unaffected female is the carrier.[33-36]

These patients are phenotypically females and have no somatic anomalies. Their stature is normal and growth potential is uncompromised. The diagnosis is made when ovarian failure is documented and a karyotype performed. Pubertal development is minimal or absent.

Since familial gonadal dysgenesis is not associated with an abnormal karyotype, the diagnosis cannot be made prenatally, but if the fetal karyotype is normal and different from that affecting the family, the infant is unlikely to be affected.

Gonadal removal is mandatory in all patients with a Y chromosome. These patients may develop a gonadoblastoma or malignant germ cell tumors such as a dysgerminoma. The risk of malignancy in dysgenetic gonads in the presence of a Y chromosome is 25%.[37,38]

Swyer Syndrome

Swyer first reported two cases of XY females with uterus and bilateral dysgenetic testes in 1955.[39] The syndrome results from testicular failure between post-fertilization days 43 and 59 of embryonic life, which is prior to the time of MIF production. The affected individuals have normal female external and internal genitalia but have a 46XY karyotype. The gonads are replaced by fibrous bands, and the patient is seen complaining of primary amenorrhea. Pubertal changes are characterized by minimal breast development, yet pubic and axillary hair growth are unimpaired. Therapy consists of hormonal replacement, as well as laparotomy and removal of the dysgenetic gonads. The rudimentary uterus can menstruate during replacement therapy. Swyer syndrome may be inherited as a male-limited autosomal dominant or an X-linked recessive disorder, although sporadic cases are known. HY antigen is positive in some patients implying a receptor defect. In others, HY antigen is not present.[40,41]

Differentiation of Swyer syndrome from the more common types of male pseudohermaphroditism due to testicular failure depends on the presence of vagina and uterus. Obviously, it is difficult to distinguish on the basis of physical findings alone whether the gonads developed and then disappeared (primary testicular failure) or alternatively have never developed (agonadism, anorchia).

Testicular Dysgenesis and Testicular Regression

Unlike patients with true agonadism, patients with testicular dysgenesis have testicular loss between gestational days 70 and 75. Still, no gonads will be present, external genitalia will be ambiguous, and components of both mullerian and wolffian ducts are present. If on the other hand testicular loss occurs between days 75 and 84, no mullerian duct remnants will be present, the external genitalia will be ambiguous, and wolffian duct structures will be present.[42]

Anorchia

Testicular loss that occurs after 90 days post-fertilization leads to the development of rudimentary testes or the vanishing testis syndrome. The karyotype is XY, and the appearance of external genitalia is dependent on the length of exposure to androgenic stimulation.

Premature Ovarian Failure

Patients with premature ovarian failure or chromosomally competent ovarian failure have a 46XX karyotype. Thus, these patients have normal stature and none of the somatic anomalies found in patients with other forms of gonadal dysgenesis. These patients usually have secondary amenorrhea, and the diagnosis of ovarian failure may be made when FSH levels are above 50 mIU/ml.[43]

Premature ovarian failure can result from numerous conditions such as autoimmune diseases, resistant ovary syndrome, environmental factors such as infectious or infiltrative diseases of the ovary, etc. Sibships with several affected individuals and paternal consanguinity would favor autosomal recessive inheritance, the 46XX pure gonadal dysgenesis, though there are reports of sporadic occurrences. Neuroauditory deficits have been seen in many of these patients. Hereditary diseases associated with ovarian failure include myotonic dystrophy, galactosemia, and mucopolysaccharidoses. Numerous infectious diseases have been associated with premature ovarian failure. These include mumps, rubella, tuberculosis, schistosomiasis, sarcoidosis, and actinomycosis.[44,45]

Receptor Deficiency

In Savage syndrome (resistant ovary syndrome) the ovary has the normal numbers of primordial follicles, but they fail to mature.[46] It is thought that these patients either lack FSH receptors or that these receptors are defective, and thus the primordial follicle is unable to respond to gonadotropin stimulation. Pregnancy has been reported in some patients with the Savage syndrome after estrogen replacement therapy or following high dose HMG treatment.[47] These patients should be treated with estrogens to prevent the development of osteoporosis, and to maintain secondary sexual characteristics.

Persistent Mullerian Duct Syndrome

The persistent mullerian duct syndrome, or as it is sometimes called, inguinal uterine hernia, affects only what appear to be normal males. There seems to be an isolated defect in the production of or a lack of response to the MIF. Therefore, the development of the fallopian tubes, uterus, and upper vagina is not inhibited in these affected males. The testes are normal, the external genitalia develop normally, and they virilize at puberty. The child usually has an inguinal hernia in which a small uterus and fallopian tubes are found. Cryptorchidism is common, and it may be the reason that in about 5% of these males a seminoma or other germ cell tumor may develop. In addition, many of these men are infertile, due either to the cryptorchidism or to damage to the vas deferens at the time of surgery. This syndrome is inherited as an X-linked recessive or a sex-limited autosomal dominant gene.[48]

Management

A newborn infant with genital ambiguity represents a medical emergency. With appropriate management, the child can have a well-adjusted life, but without treatment the child may become outcast. This newborn requires that a correct diagnosis be determined in rapid and systematic fashion. At the same time, the emotional reaction of the parents must be considered and appropriate counseling given. Both cytogenetic and endocrine studies should be initiated at once. It is crucial to exclude the syndrome of congenital adrenal hyperplasia, the most common cause of genital ambiguity, since if this is the diagnosis, the child may be a salt loser and may die during the neonatal period or soon afterwards. During the next 72 to 96 hours required for these tests, the emotional status of the parents must be considered. It is extremely important that the physician should attach no unusual significance to the occurrence of the genital ambiguity. One should emphasize that with corrective surgery, the external genitalia should appear normal and conform to the sex of rearing that was chosen. In addition, the child can expect normal psychosexual development. Regardless of the sex chosen, the parents and family should know that the child is a female or a male and nothing in between; that all contradictory sexual features have been removed or would be removed in the future; that the gonads, if of the opposite sex, should be removed, and hormonal replacement therapy be given at the time of puberty; that sex life can be expected to be normal; and that future fertility may be affected by this condition.

The XY female may escape diagnosis until puberty, at which time she fails to menstruate. At this point, the psychological development of the pubertal girl is of utmost importance. Surgery to elongate a short vagina may be necessary if graduated vaginal dilatation fails. Clitoral reduction and cyclic hormone replacement may be required to achieve feminization and prevent osteoporosis.[49] The patient must also be informed at an appropriate time that she may be infertile.

Because intersex disorders are inherited, and thus can affect other siblings, the physician must be aware of the modes of inheritance and be able to predict the risk that the mother will have another affected infant should she become pregnant again. The physician must have a working knowledge of the embryonic development of the genital tract, the genetics of inheritance, and the medical and surgical treatment of such patients before such patients can be properly managed.

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Maternal Serum Alpha-Fetoprotein Screening in Upper East Tennessee

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The Regional Clinical Genetics Center at the East Tennessee State University College of Medicine initiated a maternal serum alpha-fetoprotein screening program in October 1985. This program carefully integrated the services of a local genetics service laboratory, clinical geneticists, and perinatologists in order to offer comprehensive testing, counseling, and patient evaluations. We report here some results of the first complete year of this program.

Alpha-fetoprotein (AFP) is a serum glycoprotein with a molecular weight of approximately 70,000 daltons. AFP was discovered to be unique to the fetal circulation by Bergstrand[1] in 1956, and is produced mainly in the fetal liver. AFP is found normally in amniotic fluid and apparently diffuses through the placenta into the maternal circulation. Any defect that allows increased leakage of fetal plasma into the amniotic fluid, such as a neural tube defect (NTD) or ventral wall defect (VWD), may result in an abnormally high concentration of AFP, which will correspondingly result in a higher concentration in the maternal serum. Amniotic fluid AFP levels peak at about 13 weeks of gestation and then begin to decline. Maternal serum AFP levels, however, rise throughout the pregnancy. Appropriate interpretation of the clinical significance of individual determinations requires careful adjustments allowing for exact gestational age, maternal weight, maternal age, and the presence of maternal insulin-dependent diabetes, utilizing locally derived standards appropriate for the specific population being studied.

Beginning in the early 1970s, studies were undertaken to determine the validity of measuring AFP levels in amniotic fluid and maternal serum.[2-6] These studies were done using electrophoresis and/or radioimmunoassays, and confirmed that maternal serum AFP could be used in the detection of open NTDs. In the United Kingdom maternal serum AFP screening programs have been in use since the mid-1970s to help identify women with an increased risk of carrying a fetus with an open NTD.[7] Since the issuance of a legal alert by the American College of Obstetrics and Gynecology early in 1985, the frequency of testing in the United States has significantly increased, such that the routine offering of such testing might now be considered a fundamental standard of obstetrical care.

Materials and Methods

Approximately 5 to 10 ml of clotted maternal blood were obtained from patients who had completed 15 to 21 weeks of gestation. The preferred time was 16 completed weeks, since there is maximal separation of normal and abnormal values at that time. The specimens were either mailed to the laboratory or delivered by courier. The samples are very stable, and do not require special handling.

The AFP analyses were performed twice weekly, using an FDA-approved ELISA test developed by Abbott Laboratories. This test is a solid enzyme immunoassay based on the sandwich principle. Color intensity is read using a Quantum II spectrophotometer at a wavelength of 492 nm. All samples are run in duplicate and compared with both kit and local standards. The amounts of AFP are then reported both as absolute amounts in ng/ml and as multiples of a locally derived median (MoM). These last values are then corrected for maternal weight and for the presence of insulin-dependent diabetes. Values over 2.5 MoM or below 0.5 MoM are considered potentially abnormal.

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Basic Protocol for Management: Values between 0.5 and 2.5 MoM are reported as normal and hard copy reports are sent to the referring physician by mail the same day. When a result is over 2.5 MoM but less than 3.5 MoM at no greater than 17 weeks of gestation, a second specimen is requested by calling the referring physician's office. In instances where the value is greater than 3.5 MoM on an initial specimen, or greater than 2.5 MoM on a second specimen, or when the pregnancy is beyond 18 weeks, the clinical geneticist calls the referring physician to discuss management, which usually involves referral to the program for ultrasound evaluation, genetic counseling, and amniocentesis when no obvious explanation for the elevated value has been found. Low values are age-adjusted to determine an empiric risk for Down syndrome, and when such risk is determined to exceed 1:200 an amniocentesis is recommended.

Results

The results of the first year of screening are presented in Fig. 1. A total of 1,250 pregnancies were evaluated by the program. Several other pregnancies with abnormalities detected initially by ultrasound that were found to also have elevated maternal serum AFP levels are not included.

Using a cut-off level of 2.5 MoM for singleton pregnancies, 53 were reported as having high values. This positive rate of only 4.2% was lower than expected, and reflects the relatively high frequency of early ultrasound dating of pregnancies being done in the region, so that essentially no abnormal values were ascribed to inappropriate dates or to unanticipated twin pregnancies. On the other hand, all instances of anen-

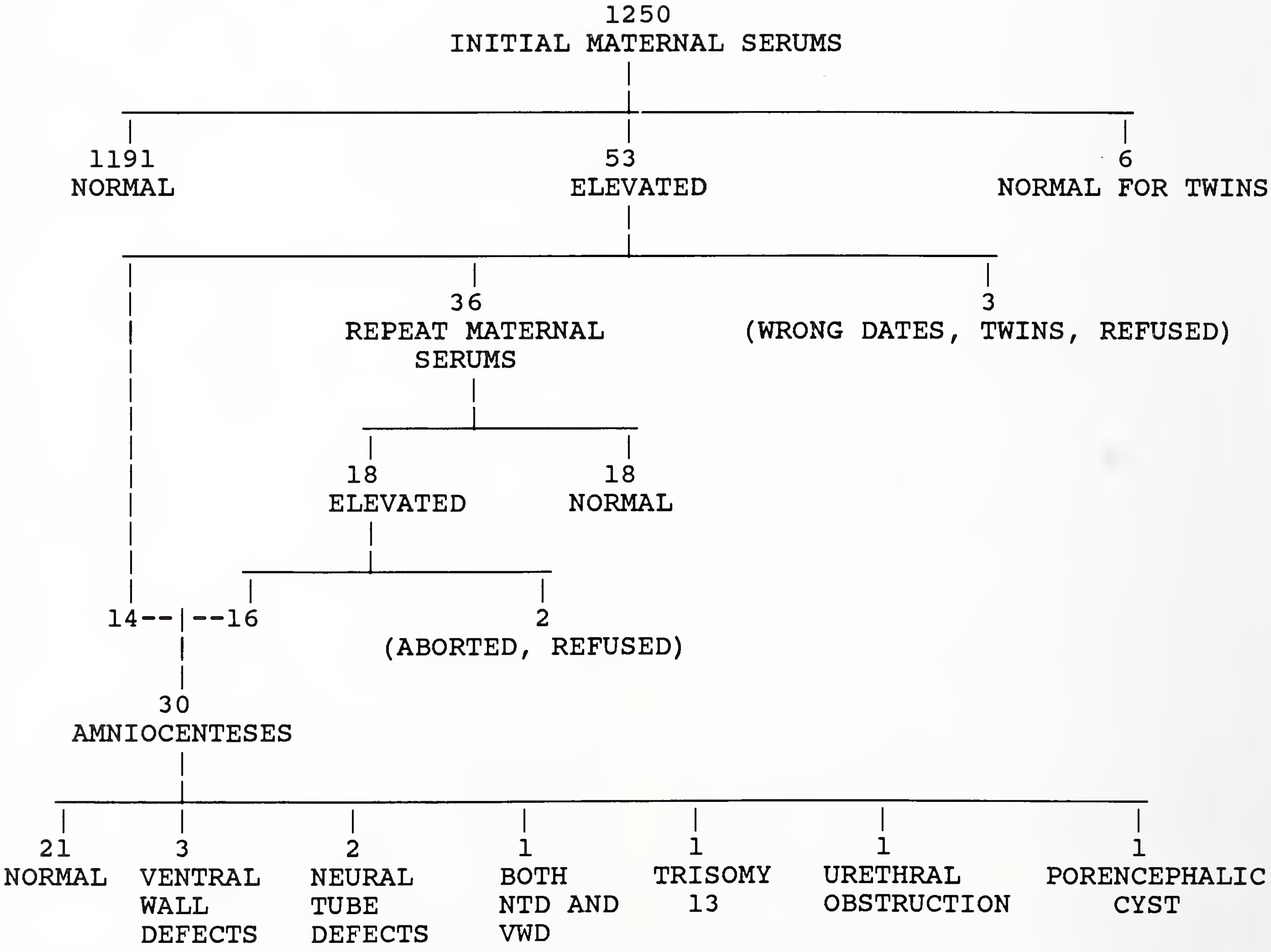

Figure 1. Maternal serum alpha-fetoprotein outcomes.

cephaly were detected through early ultrasound studies and only confirmed by AFP and acetylcholinesterase testing.

Second serum samples were obtained on 36 women; 18 of these were reported as normal. All of these originally had only marginally elevated values, and no women with initial values above 3.0 MoM were found to be normal on repeat testing. Fourteen individuals went directly to amniocentesis.

Thirty amniocenteses were undertaken, generally during the 17th gestational week. No abnormalities were noted either at ultrasound or by amniotic fluid testing for AFP levels or karyotype in 21 cases. In the nine remaining cases there were severely abnormal fetuses, including two with a meningomyelocele, three with ventral wall defects, one with both ventral wall and NTDs, one with Trisomy 13 with cyclopia, one with urethral obstruction with dysplastic kidneys, and one with a large porencephalic cyst. Fluids from the open NTDs were also found to be acetylcholinesterase-positive. All diagnoses were confirmed at delivery.

While many of the women tested are still pregnant, no false-negatives of appropriately obtained specimens are yet known.

Summary and Conclusions

Random maternal AFP screening of 1,250 women detected nine significant fetal abnormalities. Through close cooperation of the clinical geneticists and the perinatologists it was possible in all instances to obtain second maternal serums, diagnostic ultrasound studies, amniotic fluid AFP levels, genetic counseling, and any indicated interventions, generally within 14 days of initiating the first screening sample. At present this screening procedure, when carried out in such a cooperative program, appears to represent the most efficient and cost-effective prenatal screening presently in use for any type of birth defect.

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THE STATE-BY-STATE ASSOCIATION DUES LEVELS FOR 1987
(As furnished by the AMA Division of Membership)

Alabama	$250	Kentucky	$400	Ohio	$295
Alaska	400	Louisiana	235	Oklahoma	210
Arizona	375	Maine	375	Oregon	325
Arkansas	300	Maryland	255	Pennsylvania	300
California	340	Massachusetts	260	Puerto Rico	220
Colorado	555	Michigan	290	Rhode Island	400
Connecticut	275	Minnesota	400	South Carolina	220
Delaware	205	Mississippi	235	South Dakota	350
DC	615	Missouri	300	**Tennessee**	**220***
Florida	300	Montana	370	Texas	235
Georgia	335	Nebraska	285	Utah	285
Hawaii	505	Nevada	180	Vermont	225
Idaho	394	New Hampshire	275	Virginia	300
Illinois	273	New Jersey	330	Washington	398
Indiana	235	New Mexico	250	West Virginia	300
Iowa	300	New York	275	Wisconsin	465
Kansas	220	North Carolina	350	Wyoming	320
		North Dakota	270		

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Brief Summary

INDICATIONS AND USAGE: Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psychosocial difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

First Episodes (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

Recurrent Episodes:

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

CONTRAINDICATIONS: Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

WARNINGS: Zovirax Capsules are intended for oral ingestion only.

PRECAUTIONS: General: Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

Drug Interactions: Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the F_1 generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

Pregnancy: ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS — Short-Term Administration: The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

Long-Term Administration: The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

Chronic suppressive therapy for recurrent disease: One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

Intermittent Therapy: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

Patients With Acute or Chronic Renal Impairment: One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance ≤ 10 ml/min/1.73/m^2.

HOW SUPPLIED: Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200"- Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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[*] In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

Wellcome
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Research Triangle Park
North Carolina 27709

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VUH Morning Report

Histoplasmosis in AIDS

Case Report

A 34-year-old white man was admitted for the evaluation of dyspnea. The patient had felt well until two weeks earlier when he noted the insidious onset of shortness of breath and nonproductive cough. Treatment with ampicillin by his local physician had failed to improve his symptoms. Two days prior to admission he had fever to 104°F and shaking chills. His past medical history was unremarkable. He had once used intravenous drugs, but had not for several years. He denied homosexual contacts or blood transfusion. Review of systems revealed a 20-lb weight loss in the past three months.

On physical examination, the patient was well developed and well nourished and in moderate respiratory distress. His oral temperature was 101°F with a pulse of 100/min, and respiratory rate of 24/min. There was no rash or lymphadenopathy. Soft rales were heard diffusely throughout both lung fields. Cardiac and abdominal examinations were negative. Laboratory examination showed a white blood cell count of 9,500/cu mm, and a hematocrit of 35%. Room air arterial blood gases revealed marked hypoxemia, with a Po_2 of 55 mm Hg, and a moderate respiratory alkalosis. Chest x-ray revealed diffuse reticulonodular and alveolar densities in all lung fields and minimal cardiomegaly (Fig. 1).

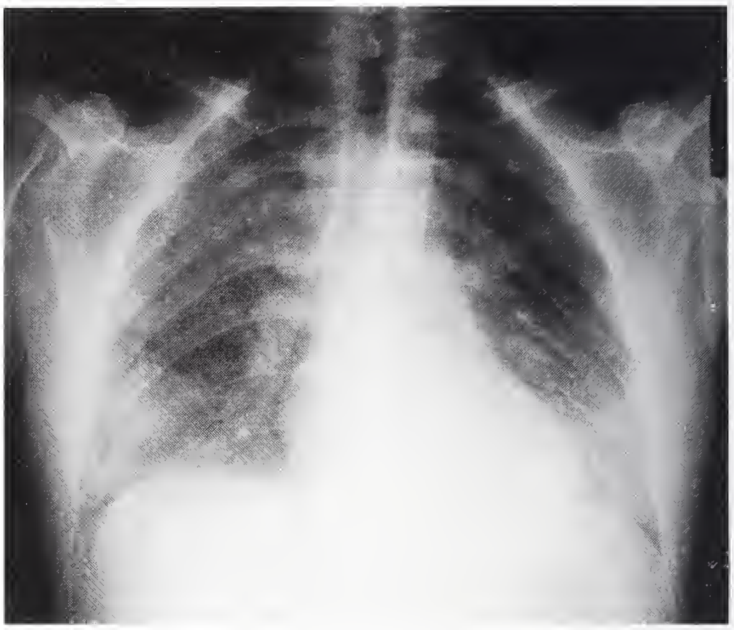

Figure 1. Portable anterior-posterior chest radiograph demonstrating diffuse alveolar and interstitial infiltrates in both lungs.

Treatment was begun using intravenous erythromycin, gentamicin, and trimethoprim/sulfamethoxazole (TMP/SMX). Flexible fiberoptic bronchoscopy with transbronchial biopsy was performed revealing characteristic forms of *Pneumocystis carinii*. Empiric antibiotic therapy was continued while awaiting culture results of the bronchial washings. The patient's condition deteriorated and he required mechanical ventilation to maintain adequate oxygenation. Amphotericin B was added to his therapeutic regimen empirically. Results of a serum test for human immunodeficiency virus (HIV) were positive and there was a reversal for the normal T_4/T_8 lymphocyte ratio. The patient's condition improved on amphotericin B and TMP/SMX and he was weaned from the ventilator. Over the next four weeks his respiratory status again deteriorated, and in accordance with his wishes mechanical ventilation was not reinstituted. He died, and an autopsy revealed diffuse pulmonary involvement with *P. carinii, Histoplasma capsulatum*, and cytomegalovirus.

Discussion

Except following heavy exposures, disseminated histoplasmosis is a rare infection, even in the immunocompromised host. The most common site of infection in patients with AIDS is the lung. These patients frequently have one of the three most common lung infections: *P. carinii*, cytomegalovirus, or atypical mycobacterial infection, particularly *Mycobacterium avium-intracellulare.*[1] Recently, several reports have emphasized the importance of specific geographic risk factors for acquiring certain opportunistic infections. Coccidioidomycosis has become a frequent pulmonary pathogen in AIDS patients residing in southern California, and histoplasmosis has recently been reported as a common pathogen in AIDS patients residing in the Ohio valley and southeastern United States.[2-4]

As in this patient, pulmonary infections are frequently due to multiple organisms, and in a patient with a deteriorating clinical course the discovery of one pathogen should not discourage the search for others. The outcome of patients with disseminated histoplasmosis has been poor. Essentially all patients with histoplasmosis have relapsed after antifungal therapy is discontinued.[3] Initial therapy with amphotericin B followed by long-term administration of ketoconazole has frequently suppressed but rarely cured this infection. Because of the rarity of disseminated histoplasmosis in normal hosts, a diagnosis of AIDS should be considered in patients with disseminated histoplasmosis in the presence of known risk factors.

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Prepared by Arthur P. Wheeler, M.D., Hugh J. Morgan chief medical resident, Vanderbilt Medical Center, Nashville.

Mannitol Intoxication

Case Report

A 42-year-old white woman with mild chronic renal insufficiency (creatinine 2.1 mg/dl), a 15-year history of non-insulin dependent diabetes, and hypertension was admitted for the treatment of acute angle closure glaucoma. Chronic therapy with an oral hypoglycemic agent and clonidine had produced good control of serum glucose and blood pressure respectively. Initially, she was treated with acetazolamide orally and 250 ml of 20% mannitol given intravenously every eight hours. On the second hospital day she underwent cyclocryotherapy for treatment of her glaucoma. Twenty-four hours postoperatively she became confused, tachypneic, and oliguric and was then transferred to the medical service. Her blood pressure was 152/100 mm Hg with a pulse of 90/min, and respiratory rate 20/min. She was oriented but lethargic. Except for fundoscopic changes of diabetic retinopathy her neurologic examination was normal. Auscultation of the chest revealed rales in the lower third of both lung fields. An S_3 gallop was present on cardiac examination. Jugular venous distention was noted with the patient at 45°, but there was no peripheral edema.

When a bedside test showed the blood glucose to be less than 40 mg/dl, D50W was given, but without improvement in mental status. Laboratory testing revealed a serum sodium of 112 mEq/L, a bicarbonate of 12 mmole/L, an anion gap of 18, and a serum creatinine of 5.9 mg/dl. Measured serum osmolality was 307 mOsm/kg H_2O, with an osmolar gap of 70. Arterial blood gases revealed a pH of 7.34, a Pco_2 of 34 mm Hg and a Po_2 of 90 mm Hg. The packed cell volume fell from 32% on admission to 26%. Chest x-ray confirmed the physical examination findings of pulmonary edema, and the patient was transferred to the intensive care unit where she was given furosemide in an unsuccessful attempt to induce diuresis. Hemodialysis was instituted for the removal of mannitol. The patient recovered full renal function within two weeks and was discharged with normal electrolytes and a serum creatinine of 1.8 mg/dl.

Discussion

Mannitol is a six carbon alcohol widely used as an osmotic diuretic to reduce cerebral edema and for the treatment of acute glaucoma.[1] When administered intravenously, mannitol cannot move across cell membranes and remains in equal concentrations in plasma and interstitial fluid. Because of its potent osmotic activity, a shift in water from the intracellular to extracellular space occurs, leading to cellular dehydration. In patients with normal renal function, 80% of a mannitol dose is rapidly excreted unchanged by the kidney, with the remainder undergoing slow hepatic metabolism and biliary excretion.[2] Renal insufficiency, even of a mild degree, significantly impairs the clearance of mannitol, leading to a marked expansion of the extracellular volume. As seen in our patient, fluid overload is common, often resulting in congestive heart failure and pulmonary edema. Lethargy and confusion are also common as a result of dehydration of brain tissue. The electrolyte disturbances seen in this patient are classic, including hypertonic hyponatremia and an osmolar gap produced by high serum concentrations of mannitol.[3] The movement of free water into the extracellular compartment produces a "dilutional" acidosis by acutely expanding the circulating volume without increasing the extracellular bicarbonate. Anemia is common in mannitol intoxication as a result of the expanded circulating volume, and the osmotic effect producing a reduced red blood cell volume. Although acute oliguric renal failure has occasionally been treated with mannitol, recent reports suggest that mannitol may actually produce renal failure in some patients.[4]

The treatment of mannitol intoxication, particularly in the presence of renal insufficiency, is hemodialysis, which can decrease the serum half-life of mannitol in renal failure from 36 to 6 hours.[5] Additionally, dialysis is useful for the treatment of the associated electrolyte disorders and symptoms of uremia. In the presence of compromised renal function mannitol doses should be reduced by approximately 1% for each 1 ml/min decrease in calculated creatinine clearance. That is, a patient with a calculated creatinine clearance of 50 ml/min should receive no more than 50% of the dose that would be given to a similar patient with normal renal function.

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Tracheobronchial Disruption

BRIAN H. GARBER, M.D.; HENRY S. NELSON, JR., M.D.; and
WALTER H. BROTT, M.D.

Tracheobronchial disruption is a devastating injury most commonly associated with blunt thoracic injury, though obviously it can be incurred with penetrating injury. There are frequently associated injuries, including some that may give clues to the diagnosis. As with other injuries due to trauma, results are maximized by early diagnosis and treatment. The following case demonstrates some of the salient aspects of such injuries.

Case Report

In a single vehicle accident, in which the vehicle went through a fence, a fence post was driven through the windshield and into the right upper chest of a 20-year-old white man, causing a blunt injury to the thorax with superficial lacerations to the overlying skin. After he was initially treated at Sweetwater Hospital with intravenous fluids, antibiotics, and a right thoracostomy tube, he was transferred to the University of Tennessee Medical Center at Knoxville via Lifestar. His trauma score on arrival was 13 and vital signs were blood pressure 176/80 mm Hg, pulse 114/min, respirations 40/min. Examination revealed a chest tube in place with a high volume air leak, and a flail segment with instability of the right upper chest wall and clavicle. The neck was swollen but nontender, and there was subcutaneous emphysema of the neck, chest, and abdomen. He was in moderate respiratory distress; the initial PaO_2 in Sweetwater was 42 mm Hg and the PaO_2 on 100% O_2 on arrival was 59 mm Hg. Oxygenation improved with adequate ventilation of the left lung through a bronchocath.

Arteriography showed no injury to the major vessels, but fiberoptic bronchoscopy and esophagoscopy demonstrated an extensive tracheal injury, with mucosal tears in the esophagus at the cricopharyngeus and at the esophagogastric junction. Thoracotomy revealed a large defect in the right mainstem bronchus, with near total avulsion of the right upper lobe bronchus. There was extensive direct damage to the right upper lobe as well. Further, there was a second rent in the posterior, membranous portion of the trachea extending from above the carina to the thoracic inlet.

A right upper lobectomy was performed, the defect in the right mainstem bronchus was closed using the membranous bronchus that was still in continuity at the site of injury, and the posterior tracheal rent was repaired primarily. The abdomen was explored and a gastrostomy tube inserted in order to avoid the need for a nasogastric tube. The patient was extubated within the first 24 hours, and the chest tube was removed on the tenth postoperative day. Follow-up bronchoscopy has confirmed good healing of the repair and no tracheal stenosis.

Discussion

Most patients with tracheobronchial disruption have other injuries that must be attended to in a rapid and orderly fashion. Airway control may be obtained with either intubation or tracheostomy, taking care that procedure does not precipitate acute airway obstruction. Tube thoracostomy, intravenous fluid resuscitation, and examination for abdominal, neurosurgical, and orthopedic injuries must be carried out as indicated. In searching for transbronchial disruption, primary presenting signs and symptoms include dyspnea, subcutaneous emphysema, pneumomediastinum, pneumothorax, and hemoptysis.[1] Disruption in the right mainstem bronchus or the distal left bronchial tree usually produces an ipsilateral pneumothorax. Tracheal and left mainstem tears produce pneumomediastinum, possibly with cervical subcutaneous emphysema. In particular, a large air leak that continues after tube thoracostomy is highly suggestive of tracheobronchial disruption. In addition, there are almost always other injuries. In a series reported by Jones et al,[1] the most commonly associated findings were major intrathoracic injuries, closed head injuries, and skeletal fractures. Surprisingly, in reported series and case reports, there is a low incidence of concomitant rib fractures. Once the injury is considered, it can usually be confirmed by tracheobronchoscopy, which is essential for giving exact direction to the operative repair.[2]

(Continued on page 226)

From the Department of Surgery, University of Tennessee Medical Center at Knoxville.

The Tennessee Newborn Hemoglobinopathy Screening Program

SUSAN ERICKSON, R.N., M.P.H. and J. CLINTON SMITH, M.D., M.P.H.

The Tennessee Department of Health and Environment has been awarded a federal grant to initiate a statewide Newborn Hemoglobinopathy Screening Program. The primary goal of this program is to reduce infant mortality and morbidity due to sickle cell disease and other hemoglobinopathies among affected individuals in the state. A secondary program focus is the identification of carrier parents and "at risk" siblings who may benefit from educational programs and reproductive counseling. We describe the rationale for initiating this screening program, the multi-institutional collaboration required for development and implementation, and the projected benefit to the citizens of Tennessee.

Reducing Infant Mortality/Morbidity Due to Sickle Cell Disease

Recent data emphasize that detection and treatment of hemoglobinopathies in the newborn period can reduce childhood mortality due to this disease.[1] It has been well documented that children with sickle cell anemia have increased susceptibility to bacterial infections, particularly those caused by *Streptococcus pneumoniae*. Pneumococcal meningitis, septicemia, and pneumonia remain the major causes of death in affected children, with those under 3 years of age at greatest risk of fatal infections.[1-3] Efforts at reducing mortality and morbidity by early diagnosis, close medical supervision, and prompt antibiotic therapy at the onset of symptoms have not been consistently effective.[1] The subtle onset and fulminant character of infections in these children limits the effectiveness of this approach and necessitates a plan for prevention.

In 1983, a clinical trial was initiated at the Sickle Cell Disease Branch, National Heart, Lung and Blood Institute of the National Institutes of Health to test the effectiveness of oral penicillin prophylaxis in reducing the incidence of bacterial infection in children with sickle cell disease. This randomized, double-blind, multicenter, and placebo-controlled study noted an 84% reduction in the incidence of *S. pneumoniae* septicemia, and no deaths in the penicillin-treated group. These initial data were conclusive enough to warrant early termination of the study. The Sickle Cell Disease Branch of the NIH now stresses the importance of newborn screening programs to assure that all infants with sickle cell disease are diagnosed early and antibiotic prophylaxis is initiated by 4 months of age.[1] Congress has now authorized in the 1986 Omnibus Budget Reconciliation Act that money be set aside for projects to screen newborns for sickle cell anemia and other genetic diseases.

Identification of Carrier Parents and At Risk Siblings

The identification of carrier parents and trait or disease affected siblings through screening newborn infants has been referred to as "the benefit beyond the target."[4] These family members, once identified, can be referred for education and reproductive counseling. The ability to retrospectively identify carriers is a secondary advantage of screening newborns for hemoglobinopathies.

Program Design

The Tennessee Newborn Hemoglobinopathy Screening Program was developed through the collaborative efforts of Meharry Medical College Sickle Cell Center, the Memphis Regional Sickle

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From the Maternal and Child Section, Tennessee Department of Health and Environment, Nashville.

Cell Center, and the Maternal and Child Section and Bureau of Laboratories of the Tennessee Department of Health and Environment.

Participation in this program will be voluntary. All parents of newborns will be given the opportunity to be screened, but parents of nonwhite infants will be targeted for participation. Twenty-five hospitals in Tennessee that together report 91.4% of the nonwhite births will be asked to participate in the first phase of program implementation. The hemoglobinopathy screen will use the same filter paper blood sample currently collected for phenylketonuria and hypothyroidism screening of all newborns.

Notification of negative results will be sent via mail to the infant's health care provider. Positive screening results will be processed by the Maternal and Child Health Laboratory coordinator. The provider and local health department will be notified and instructed to obtain a liquid blood sample for definitive diagnosis at the program reference laboratory, the Meharry Sickle Cell Center. Test results will be reported to the laboratory coordinator, who will notify the appropriate provider, the local health department, and the hemoglobinopathy program screening coordinator.

A database will be maintained by the newborn hemoglobinopathy screening program coordinator, and will provide the basis for evaluating program effectiveness. This database will also help eliminate the costly and unnecessary retesting that often occurs in screening efforts in this state. Communication and cooperation between providers and the screening program coordinator will be essential to assure that program objectives are met.

Program Impact

Birth data for 1985 show a total of 71,808 live births in Tennessee, with 16,805 nonwhite births (about 24%). Assuming an incidence of sickle cell disease in one out of every 525 nonwhite live births, it is anticipated that approximately 32 infants a year will be born with hemoglobin SS disease. If one includes other symptomatic hemoglobinopathies, such as hemoglobin SC and hemoglobin S/beta thalassemia, about 40 to 50 infants will be identified annually in Tennessee who are in immediate need of medical attention.

It is estimated that one of every ten nonwhite infants born will have sickle cell trait (hemoglobin AS). Again, based on 1985 data, estimates project that between 1,600 and 1,700 infants will be born with this trait each year in Tennessee. The parents and siblings of these identified newborns will need counseling, education, and often evaluation.

Since this program is *voluntary* and not mandated, the figures projected above may vary with participation level. A coordinated educational effort will focus on enhancing community awareness and utilization of the screening program. Since Tennessee ranks in the quintile of states with the highest infant mortality rate (13.1 deaths/1,000 live births—1984 recorded data, by place of occurrence), it is logical and expedient to pursue those programs that will improve the longevity and health of our citizens, and bring our infant mortality rate closer to the national average. The support and cooperation of the Tennessee medical community will be essential for the success of this program.

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It Looked Sooo Simple!

J. KELLEY AVERY, M.D.

Case Report

On May 5, a 30-year-old male school teacher reported to a board-eligible internist with a history of a "lump" in the region of his right groin. This was the first visit by this patient to this physician. Examination of the patient resulted in the following notation in the office records, "Nodule, right groin, 2-3 cm, slight tender, not reducible (illegible, illegible, illegible) ulcers." The preliminary impression was "lymphadenitis." Prescriptions were given for Motrin, 600 mg three times a day, and Vibramycin, 100 mg twice a day.

The next visit was ten days later, when the physician noted in the record, "no improvement ?lymphoma removed, closed with 3-0 chromic—drain (illegible, illegible, illegible)." A subsequent note, dated the next day, states "lymph nodes, showing both acute and chronic inflammation."

However, on the same day of the surgery, the patient was admitted to a local hospital by the attending physician. The medical record there reveals that the patient had fainted on attempting to leave the physician's office and he was brought to the emergency room. The hospital record indicated a past history of his being a "free-bleeder." Physical examination revealed a large hematoma at the site of the surgery. Treatment was bed rest, observation, and conservative measures, i.e., heat, antibiotics, etc. No further bleeding was noted at the site. Admission PCV was 42. The day after admission the PCV was 39.2. No further change in the operative site was noted in the record and he was discharged on the third hospital day. The discharge note stated "modest bleeding." "Patient wishes to go home, cautioned to stay at bed rest, will call tomorrow." On the day of discharge, the PCV was recorded in the hospital record as 37.7. Two days after discharge the patient was admitted to another hospital where the wound was explored. A large infected hematoma was evacuated and a small arterial bleeder was ligated. Culture of the wound grew *Staphylococcus aureus,* resistant to penicillin. The wound was left open and subsequently healed with moderate scarring.

The patient was able to return to work six weeks after the initial surgery. No formal pathology report of the lymph node biopsy was ever recovered. Litigation followed.

Loss Prevention Notes

When a bad result occurs and litigation has begun, we succeed or fail on the basis of our own medical records. In this case, there were not only significant places in the record that were totally illegible, but documentation of important facts was lacking. There was no record in the initial history of the patient's being a "free-bleeder." The patient had been placed on a nonsteroidal, anti-inflammatory agent which is known to reduce clotting ability. The description of the findings at the time of the biopsy was practically nonexistent. Again, there were words that were totally illegible. Although one of the preoperative diagnostic possibilities was "lymphadenitis," no culture was done at the time of the biopsy. The hospital record showed a significant drop in the PCV from the time of admission to the time of discharge three days later. This was obviously ignored by the attending physician or considered to be of no significance. In spite of ample evidence of blood loss at the operative site, the physician did not elect to explore the area in an attempt to stop the bleeding or, better yet, ask for a consultation from a more experienced surgeon.

There is another extremely important question in this case: did the physician, a board-eligible internist, have appropriate credentials to do what he did? Although all physicians are licensed by the State of Tennessee to do "medicine and surgery," the attending physician in this case did not appear to have been trained to do a biopsy of this type. In all probability, had he recognized the significant blood loss to be a potential hazard, as it was, he would have asked for a consultation immediately after admission to the hospital on the first occasion, and his vulnerability to a charge of negligence would have been considerably less.

In reviewing the medical records of physicians involved in litigation, we frequently see a note similar to the note that appeared prior to the discharge of the patient. The note stated, "patient wishes to go home." In this case, even though the patient was anxious to go home, the hematocrits indicated that a significant amount of blood

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had been lost into the operative site and appropriate action would have been to deal with that complication in the hospital. Many times in the treatment of our patients we simply cannot allow the patient's wishes to override our better medical judgment.

It goes without saying that the presence of incomplete and illegible records, the ignored significant laboratory findings, the lack of appropriate consultation, and the significant questions as to our physician's training and experience to do this type of surgery, all contributed to a virtually indefensible case and a significant settlement was made to both the patient and his spouse.

Trauma Rounds . . .

(Continued from page 222)

McGrath[3] experimentally demonstrated that the closed, distended trachea will burst longitudinally when submitted to a blunt blow. This is what would be expected from the mechanics of bursting cylinders. The injuries noted in this patient were probably the result of both direct injury to the right upper lobe and a longitudinal bursting of the trachea from sudden compression of the chest with the glottis closed.

The key to successful results is early treatment by primary repair, first performed by Scannell, of Boston.[4] In the immediate post-injury period the trachea may remain patent because it is a fairly rigid cartilaginous structure, but in time granulation tissue will result in stenosis and subsequent functional compromise. Stenotic areas can sometimes be treated by dilatation. Rarely, injuries of the proximal mainstem bronchi may necessitate pneumonectomy. Long-term results may be quite satisfactory. The most frequent long-term complication noted by Jones et al[1] was speech impairment secondary to direct vocal cord injury or recurrent nerve injury.

Tracheobronchial disruption is an unusual and easily missed injury of increasing frequency in our mobile society. It is associated with the multiply injured patient. There are, however, keys to the early diagnosis, and thus to early treatment and optimal results.

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Send to: TMA, 112 Louise Ave., Nashville, TN 37203

JAMES R. ROYAL

Bye, Bye

Last year at this time I wrote that I hoped my feet would grow to fill the shoes of my predecessors. I still wear the same size shoes, but my belt has been let out a notch—the perils of this job. It has been challenging and fun, and Pat and I wish to thank all of you for your kindness and support. Hadley and Don and all of the TMA staff have been *super* in bringing us through this unique year. I owe a special *thanks* to Jean Wishnick for helping prepare the 12 President's Pages—on time.

This year has been interesting. We have embarked on some very timely programs. The TMA-sponsored statewide IPA is in place and should be functioning shortly, thanks to a very capable and hardworking task force of our members. Even though some did not agree with its development, it is our baby sired by our House of Delegates. We should all help nourish it to maturity.

This year we joined with numerous groups to form the Tennessee Association for Civil Justice Reform. For the first time the physicians of Tennessee are not alone in their fight to bring some meaningful reform to the tort laws. All sectors of society have joined us—except of course the trial lawyers.

We are actively working with the Governor, the Legislature, and the Hospital Association to help find a solution to the problem of indigent and uncompensated care.

We have some problems with the Mid-South Foundation for Medical Care, the designated Medicare PRO for Tennessee. We have established a dialogue with their board of directors and are working toward better relations.

Medicare rules are being changed to try and force physician participation with fee freezes, reductions, and payment denials.

Nurse practitioners, anesthetists, optometrists, and chiropractors all want to practice medicine and are pressuring for legislation to allow them to do that.

So we did not solve all the problems this year. They continue to multiply and expand. Next year should be just as interesting.

Fortunately, we have a very strong and capable incoming president, Dr. James T. Galyon from Memphis. He has served the TMA many years in several leadership positions, including chairman of the Board of Trustees. He knows the job and is as qualified as anyone could be. I wish him well and urge all of you to join me in supporting him.

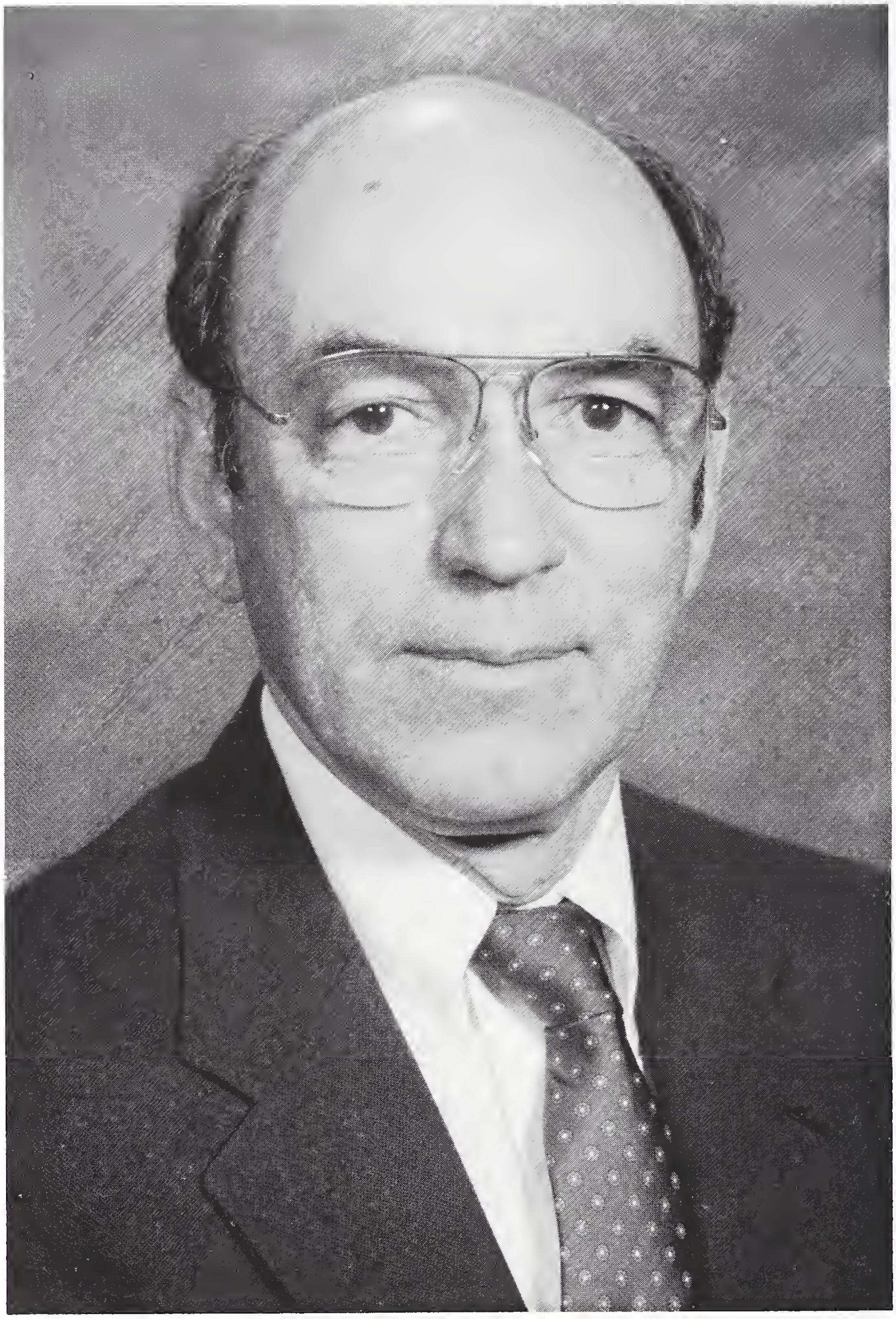
One final thought I would like to leave with you. I believe the most significant threat to our profession is not the proliferation of alternative delivery systems but changes mandated through legislation and the courts. Several provisions of the Omnibus Budget Reconciliation Act of 1986 which aim to force physicians into participation in Medicare are a preamble to what some of the politicians have in mind for us. The worst provision requires nonparticipating physicians to refund payments for services that the Medicare carriers deem to be unnecessary. We must continue to oppose such dreadful and harmful regulations.

Unlike the great American Caesar, this old past president does not intend to just fade away. I intend to stay active and help Dr. Galyon, in any way I can, to be the best TMA president ever.

Bye, Bye

James R Royal, MD

THE NEW PRESIDENT



JAMES T. GALYON, M.D.
MEMPHIS

JAMES T. GALYON, M.D.

133rd President—Tennessee Medical Association

The 133rd president of the Tennessee Medical Association, James T. Galyon, M.D., is an orthopaedic surgeon. He has maintained a private practice in Memphis since 1961.

Dr. Galyon was born in Knoxville, Tennessee, on April 13, 1930. He graduated from Knox County Central High School in 1948 and received his bachelor of science degree from the University of Tennessee Knoxville in 1950. In 1953, he received his doctor of medicine degree from the University of Tennessee College of Medicine and went on to an internship at Richmond County Hospital in Columbia, South Carolina. After serving in the U.S. Army Medical Corps in orthopaedic service in Germany from 1955 to 1957, he completed his residency in general and orthopaedic surgery at Kennedy General Hospital in Memphis. He also completed a residency at Arkansas Children's Hospital and at the University of Arkansas Medical Center in Little Rock in 1960-1961.

In 1971, Dr. Galyon served as president of the medical staff at St. Joseph Hospital in Memphis. In 1976, he was president of the medical staff at St. Francis Hospital in Memphis, and he later went on to be chairman of the hospital's Constitution and Bylaws Committee and Department of Orthopaedics in 1978.

Dr. Galyon served on the Tennessee Medical Association Board of Trustees from 1980 to 1983, and was its chairman in 1982-1983. He was also a member of the TMA ad hoc committee to study formation of a hospital medical staff section. In 1980-1981, he was president of the Memphis and Shelby County Medical Society, and he continues to serve on several of its committees.

Dr. Galyon has served as St. Francis Hospital's representative to the AMA Hospital Medical Staff Section since 1984. He has also been a member of the Tennessee Board of Licensing Health Care Facilities since 1986 and the Memphis Coalition for Health Care since 1981.

Certified by the American Board of Orthopaedic Surgery, Dr. Galyon has served as clinical assistant professor of orthopaedic surgery at the University of Tennessee Center for the Health Sciences since 1973.

Dr. Galyon has served as a member of the Presbyterian Services Board of Trustees, and has been an elder of the Second Presbyterian Church since 1980. He and his wife, Virginia, have three children.

With his professional credentials and record of service to organized medicine and his community, the Tennessee Medical Association is honored to have James T. Galyon, M.D. as its 133rd president.

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APRIL, 1987

editorials

On Finessing the Rubus

Rubus, rubus—gastric acid;
All this laughing leaves me flaccid.

On the wall in my office is a sign that reads: "NOTICE—*While in this room speak in a low, soothing tone and do not disagree with me in any manner.* Please be informed that when one has reached my age, *Noise* and *Non-concurrence* cause gastric hyperperistalsis, hypersecretion of the hydrochloric acid, and rubus of the gastric mucosa, and *I become most unpleasant!*" It is an elegantly executed sign, decorative without being gaudy, and large enough to be noticed but not so large as to be obtrusive. In other words, just right on all counts—including its message. I don't remember where I got it, or when; it has been posted there, in a succession of offices, for a long time—30 years at least. Its caveat has not been uniformly observed.

I am not certain what age limit is implied in the message, but experience has led me to believe that the calendar has little or nothing to do with it. Perhaps there was a time—maybe when the sign was concocted, though I doubt it—when one could enter his "room"—be it office, den, living room, parlor, or what have you—and prop his feet up in an atmosphere of peace, quiet, and calm without stopping up his eyes and ears, putting his brain on idle, and listening to the whir of his carotid arteries. If so, them days is gone forever.

I think I got the sign during the Korean War—certainly not a placid time. There followed the Johnson era, Vietnam, and the hippie generation—and through it all the Cold War; none of them was restful. We have had to put up with Pizroes, Hickfas, Cobras, and all sorts of intrusions by government agencies, such as the FDA, the IRS, the H_2S (DHHS) and so on. If one is awake he is hard put to escape at all, and even if he is not, rubus of the gastric mucosa can be calculated to give him nightmares.

One of my associates quotes his father, a staunch Roosevelt liberal Democrat, as having observed that if you want to live like a Republican, vote like a Democrat. There was a time not so very long ago that I would have accorded such a notion scant attention. Not anymore. Not only have physicians fared poorly under the present administration, but so have the middle class, the poor, and the elderly. The farmer has not done so well either, nor have small businesses. Big business may have done a little better. About the only group I can see as having flourished is the banks. To be fair, I am not sure things would or would not have been different under a Democratic administration, or a Republican government (which we have not had—not with Mr. Tip O'Neill around). I think it is clear that so long as the banks flourish, Washington is happy.

The squeeze on medicine is coming from two directions—government, whose Medicare program suffers from budget constraints (not enough money; it's all in the banks—or at least, it went through the banks to foreign shores, sometimes with disastrous consequences), and big business, which thinks it can't afford the health care package the unions require for their members. Hence our problems with PROs and insurance companies. It is always easy to find officious functionaries to cater to their whims; some of them sport an M.D. degree. We should also take no comfort in the medical background of the current Secretary of the DHHS. Since he was a doctor, Secretary Bowen has been a legislator for 14 years and a two term governor of Indiana. He is not one of us, but one of them.

I made my peace with that sign on my wall a long time ago. It is up there not as a caution to others, who seldom take note of it, but to myself, that noise and non-concurrence can indeed cause rubus of the gastric mucosa, which makes your stomach hurt and its mucosa bleed. I have therefore adopted a laissez-faire attitude toward things I can do nothing about. I sometimes write about them even when they don't directly affect me, but I keep the hydrochloric acid down.

Take the PRO. Its bureaucratic bungling and machinations affect me as a pathologist only insofar as they make my clinical colleagues unhappy. After all, as a "RAP," I have DRG problems I have not even used yet. So it would seem not unreasonable, I think, if I did not get too exercised over what the PRO does. Still—

The latest piece of nonsense to emanate from the Mid-South Foundation for Medical (Non) Care surpasses anything that I would have thought possible, even for it. Since I am certain my readers have all seen the latest regulations (except for the relative few who live out of state), I shall not soil these pages with a description of them. What they do, in effect, is institute a demerit system for the puerile providers of medical care that our PRO obviously thinks the doctors of Tennessee are. Item: If you give a vegetarian meat on his tray, that is a "medical mishap or mismanagement without potential for adverse outcome on the patient." (So help me, that is one of the examples given. Would I lie to you?) It is a Level I demerit, to be sure, but it is nonetheless a *mishap,* and a blot on your scorecard. If you do it ten times, you may get an initial notice of sanction.

This would be a howling comedy if it were not serious business, of which it makes a mockery. Unquestionably, the more serious infractions need scrutiny, but to throw in a bunch of inconsequential minutiae at the front end, with a score sheet to boot, entitled "Thresholds and Interventions Summation," trivializes the whole process, and makes a laughing stock of the PRO. Of course, you have to be uninvolved in order to afford a laugh—at least in public. Too, not all of those involved may think it uproariously funny. Having a perverse sense of humor, though, I chortled in glee.

By making itself appear ridiculous, the PRO has severely truncated its effectiveness; if we are to have a PRO—and according to law we are—an ineffectual one is not what we need.

After the glee, torpor sets in. Noise? Monstrous! Non-concurrence? Ponderous! But gastric rubus? It is to laugh!

J.B.T.

Shoo, Stork. Go 'Way

Watching a bull indulge himself out in the pasture, a boy casts a sidelong glance at the girl sitting beside him on the fence and says, "Gee, I sure would like to be doing that." "Go ahead," she says. "It's your cow."

This is sex education of sorts. It is the sort known as "putting ideas into their heads." People who hold this view are the ones who make all the noise at rallies opposing sex education in the public schools. They have short memories, or else they had vacant heads. Not having the ideas is not the same as controlling them. Control comes through education—*early* education, by precept.

If you lay the responsibility for sex education onto the school system, they pontificate, it takes responsibility away from the family where it belongs, thereby weakening the warp and woof of society. Agreed. But did you ever notice the demographics of home burglaries? Not many happen on skid row. It is hard to take much away from paupers. One has to wonder just how many of the conscientious objectors are doing a great job of sexually educating their own brood, and if they are, why they are so opposed to having someone—*anyone*—educate their neighbors' kids, many of whom have no real family. The fabric of society is already rent asunder; limiting sex education is not going to patch it up.

It is certainly true that a firm "No" will solve all, or at least most, of the problems related to sexual activity. They are correct insofar as that is the responsibility of the family; it is not anything that can be taught in a course—either at home, in church, or at school. It is only one result of firmly inculcated moral responsibility that has to be learned by precept, starting in the cradle. But friends, in case you don't know it, given the proper circumstances even the toughest armor has chinks that can be pierced; it has happened many, many times. It is then that sex education comes in handy—and in fact, lack of it is most unhandy, not only to the couple, but to their parents and to society as well. Those failures are the ones who help clutter the abortion clinics; before there were abortion clinics, they were the victims of criminal abortion, who cluttered Gyn clinics and wards with sepsis. Before antibiotics, they died. Memory of such tragedy fades fast. Most of those manning the picket lines never even knew about it; it was a long time ago.

Since the sap rises early—earlier than ostrich-like parents will admit—the alternative to abortion is teen—early teen—age parents; that begets such juicy news items as that after a nasty court fight a 14-year-old youth in Springfield is denied custody of his 16-month-old daughter in favor of her 17-year-old mother. Or that a 16-year-old Nashville East High School student gave birth to a baby boy in the school restroom and simply walked away, leaving the baby face up in the toilet. Neat, huh? There were 1,034 other teen-age girls in the same fix last year in Nashville alone.

According to this morning's paper, in the name of morality parent and church groups are bombarding the Legislature, lobbying against sex education, despite the fact that even if their young do need ideas put in their heads, which they don't, they can get more of those in a half hour of prime time TV or five minutes of daytime soap-opera than they could ever get from a year in a sex education course. The difference is that from the tube they learn all about execution but nothing about prevention—all acceleration and no brakes.

Many of the girls who get into trouble have no family to instruct them; nobody cares. The school is their only hope. Many others have families who for one reason or another do not instruct them, often because they underestimate the power of the hormones. Those children are all left to their standard youthful libidos without really understanding how babies are made—not to mention VD.

Though even the best of education on any topic may fail in the heat of the moment, it is far ahead of whatever is in second place. The medical profession's duty is to care for the sick and wounded and to prevent disease. The best answer to the abortion dilemma is to prevent unwanted pregnancies, which can be accomplished only through education. That is also the only preventive for sexually transmitted diseases. Much as we might wish it otherwise, moral suasion with the wages of sin and so on fails in the breach. One unidentified school principal was quoted in the *Nashville Banner* as saying, "The political reality is that we're not going to have sex education." We need to do our level best to see that political reality changed.

Our young ought at least to know that the stork is a myth, and that neither birds nor bees need be concerned about VD—but they do.

J.B.T.

On Feeding the Kitty

"There are," once said some wag, "a lot more people living off cancer than are dying of it." Given its current sub-epidemic status, that may not always be true of AIDS, but at least for the moment it appears close. Now that we have the diagnostic means, and have found out how many people carry the virus, the rush is on toward treatment and prevention of AIDS. No one can wait, and as usual the entrepreneurs and Madison Avenue types head the pack of predators. ICN, a small drug company in California, has made the news by having a press conference promoting its new antiviral agent Virazole, which, because it has not yet been properly tested, is also not yet approved by the Food and Drug Administration (FDA) for use. Though it appears to have promise of at least slowing development of the disease in infected individuals, the evidence is based on only a six-month trial in 16 individuals, eight treated with placebo and four each with low and high doses of the drug. At the end of the six months all of the placebo patients have developed AIDS, whereas none of the high dose group had; two months later, however, two of those also had the disease.

Whenever there is a crisis, recriminations fall down like the dew. For simply doing its job the

FDA is accused of dragging its feet, and the CDC research unit of obfuscating its findings and tampering with promising investigation, all in response to some reputed homophobic passion in the White House. I thought this a particularly appropriate subject to write about today, since it is Friday, Feb. 13, and I heard tell the first Friday the 13th every year is "Blame it on Somebody Else Day." Those who have AIDS are understandably concerned; those who know they have been exposed, and particularly those found to harbor the HTLV-III (or HLV) virus, are understandably scared. Much of the rest of the population has a bad case of AFRAIDS, mostly unjustified, since all the evidence points to lack of transmissibility by means other than sexual contact, licit or il-, or contact with infected blood. Statements to the contrary notwithstanding, that is not a fabrication of the public health authorities. The virus is fragile, and is easily killed by just about anything, including drying. Though there has been by now abundant opportunity, what with all the AIDS cases worldwide, for transmission through close non-sexual contacts, none has been documented. Nevertheless, all those groups—AIDS and AFRAIDS patients and their contacts—have put the heat on anyone heatable, thereby opening the door to opportunists.

It is particularly unfortunate when those wishing to capitalize on human misery are in positions of responsibility that offer the opportunity to do it. It is to some degree expected of the news media, even though it should not be, since that is the way they respond to everything; it is the way they keep meat on the table. If you make something a crisis, it will sell your offerings indefinitely—but just so long as it remains a crisis.

Much of the so-called AIDS crisis is media generated; certainly almost all of the AFRAIDS is. When the irresponsible party is an executive of a drug house, though, who is tooting his own illegitimate horn, it becomes offensive, not to say dangerous. Upon the premature announcement of the alleged benefits of Virazole, the drug began being procured in Mexico, where it is legally available, and smuggled into this country. Against the advice of the FDA to keep quiet until its evaluation had been completed, ICN behaved in a way that seems at best unethical.

Even more discouraging has been the news from the CDC, with accusations of tampering with the experiments of other investigators and subsequent denials and recriminations, along with accusations that work was being impeded by homophobic administrators. Those accounts have been aired not only in the lay press, but also in *Science,* the official publication of the American Association for the Advancement of Science. There was a time not so long ago when scientists were considered above such things, though the escutcheon of science has been sullied repeatedly during the past couple of decades. Since scientists are after all only people, this should astonish no one, but it always seems to. That it should not, though, makes it no less disappointing. It is sort of like finding a preacher in a bawdy house. The best that can be said for it is that it looks bad; it somehow taints the whole profession by the sort of guilt by association for which the human animal is noted.

AIDS is killing a lot of people; it is due to kill a lot more. It is also helping to keep food on a lot of tables. Without wishing harm to anyone, it is devoutly to be hoped that that particular source of nourishment will dry up—and soon, though given the present state of our ignorance, I wouldn't count on it—at least not soon. On the other hand, AFRAIDS is entirely anthropogenic and feeds on the propagation of ignorance; we can do a lot there to choke the kitty. Though some might consider that cruelty to animals, it is, to use the vernacular, "people-friendly."

J.B.T.

in memoriam

James L. Booth, age 68. Died February 11, 1987. Graduate of Tulane University School of Medicine. Member of Memphis-Shelby County Medical Society.

Malcolm Freels Cobb, Sr., age 78. Died January 28, 1987. Graduate of University of Tennessee College of Medicine. Member of Knoxville Academy of Medicine.

William Craig Grant, age 55. Died January 29, 1987. Graduate of University of Tennessee College of Medicine. Member of Memphis-Shelby County Medical Society.

John W. Griffith, Jr., age 66. Died February 7, 1987. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

Thomas Fite Paine, Jr., age 68. Died January 29, 1987. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

BLOUNT COUNTY MEDICAL SOCIETY
Cheryl E. Coleman, M.D., Maryville
Thomas E. Minnich, M.D., Maryville
Robert S. Valentine, M.D., Maryville

BRADLEY COUNTY MEDICAL SOCIETY
Elizabeth Allen Daubner, M.D., Cleveland

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY
Stuart Peter Bacon, M.D., Dayton
Joseph B. Cofer, M.D., Chattanooga
Victor A. Duncan, M.D., Chattanooga
Phillip Wesley Forsythe, M.D., Red Bank
Sahin Saban Kocacitak, M.D., Fort Oglethorpe, GA

DICKSON COUNTY MEDICAL SOCIETY
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GREENE COUNTY MEDICAL SOCIETY
William Joseph Scott, M.D., Greeneville

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Malcolm A. Cox, M.D., Savannah

MAURY COUNTY MEDICAL SOCIETY
John Frederick Worthman, M.D., Columbia

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Kenneth J. Gaines, M.D., Memphis
Ralph C. Goodman, M.D., Memphis
William Owen Hartness, M.D., Memphis
Russell E. Howard, M.D., Memphis
John Claude Morse, M.D., Memphis
Truman Franklin King, M.D., Memphis
Phillip Royal Langsdon, M.D., Memphis
Penny Beth Lynch, M.D., Memphis
Gailen D. Marshall, M.D., Memphis
Clifford James Meservy, M.D., Memphis
Robert Horace Miller, III, M.D., Memphis
S. Gail Thurmond, M.D., Memphis
Bryan Ross Troop, M.D., Memphis
Renga I. Vasu, M.D., Memphis

(Students)

James G. Avery, Memphis
Sandra M. Baltz, Memphis
Robin E. Barnett, Memphis
Brent A. Barrow, Memphis
James T. Batey, Memphis
Michael T. Beckham, Memphis
Rick Belcher, Jr., Memphis
James F. Benson, Jr., Memphis
Rusty Bernard, Memphis
John M. Bills, Jr., Memphis
Mark G. Bowles, Memphis
Edward E. Breazeale, Jr., Memphis
Jeffrey B. Brown, Memphis
James K. Buck, II, Memphis
Eldridge G. Burns, Jr., Memphis
Barry Callahan, Memphis
Robert C. Collier, Memphis
Suzanne Collier, Memphis
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J. Brian Hopper, Memphis
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R. Alan Davis, M.D., Bristol
I. N. Kutty, M.D., Kingsport
Brady B. Stoner, M.D., Bristol

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William R. Caldwell, M.D., Gallatin

WARREN COUNTY MEDICAL SOCIETY
Brian C. Beatty, M.D., McMinnville
Rene Carlos Del Valle, M.D., McMinnville

WHITE COUNTY MEDICAL SOCIETY
Robert Hoyt, M.D., Sparta

TMA Members Receive AMA Physician's Recognition Award

Twenty-one TMA members qualified for the AMA Physician's Recognition Award during January 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Elaine M. Bunick, M.D., Oak Ridge
Fenwick W. Chappell, M.D., Memphis
Thomas R. Duncan, M.D., Columbia
Clifton E. Greer, Jr., M.D., Nashville
Harvey H. Grime, M.D., Lebanon
Robert L. Haley, Jr., M.D. Madison
Hari S. Kapur, M.D., Memphis
Cecil D. Knight, M.D., Cleveland
Michel E. Kuzur, M.D., Nashville
John C. McAmis, M.D., Maryville
Amos Okrah, M.D., Memphis
Reinaldo A. Olaechea, M.D., Crossville
Richard P. Ownbey, M.D., Nashville
Arthur K. Patterson, M.D., Nashville
Daniel R. Ramey, III, M.D., Memphis
Thomas A. Russell, M.D., Memphis
Donald W. Schmidt, M.D., Memphis
Burton Silbert, M.D., Nashville
Daphine Sprouse, M.D., Nashville
John B. Thomison, M.D., Nashville
Guy Zimmerman, Jr., M.D., Byrdstown

personal news

Robert A. Beasley, M.D., Cleveland, has been inducted as a Fellow of the American Academy of Orthopaedic Surgeons.

Duane C. Budd, M.D., Johnson City, has been appointed president of the Tennessee Board of Medical Examiners.

Dennis C. Ford, M.D., Cleveland, has been certified as a Diplomate of the American Board of Anesthesiology.

Donathan M. Ivey, M.D., Crossville, has been inducted as a Fellow of the American College of Surgeons.

Kimball I. Maull, M.D., Knoxville, has been elected president of the Eastern Association for the Surgery of Trauma. Dr. Maull has also been appointed by the National Academy of Sciences to a 12-member congressional commission studying the problem of alcohol and drug use and its role in automobile accidents.

Kenneth H. Tozer, M.D., Milan, has been certified as a Diplomate of the American Board of Surgery.

announcements

CALENDAR OF MEETINGS

NATIONAL

May 1-4	American Federation for Clinical Research—Town & Country, San Diego
May 1-4	American Society of Clinical Investigation—Town & Country, San Diego
May 1-4	Association of American Physicians—Town & Country, San Diego
May 3-7	American Association of Neurological Surgeons—Dallas
May 3-7	Uniformed Services Academy of Family Physicians—Orlando, Fla.
May 4-8	Association for Research in Vision and Ophthalmology—Hyatt, Sarasota, Fla.
May 5-7	American College of Legal Medicine—Long Beach, Calif.
May 6-10	American Psychoanalytic Association—Hyatt Regency, Chicago
May 7-10	American Academy of Psychoanalysis—Chicago
May 9-13	American Society for Gastrointestinal Endoscopy—Hyatt Regency, Chicago
May 9-15	American Gastroenterological Association—Hyatt Regency, Chicago
May 9-15	American Society of Neuroradiology—New York
May 10-13	American Thoracic Society—Marriott/Sheraton, New Orleans
May 10-13	Society for Cardiovascular Anesthesiology—Marriott, Palm Desert, Calif.
May 10-14	Aerospace Medical Association—Las Vegas
May 11-13	Society for Surgery of the Alimentary Tract—Chicago
May 12-15	Society for Cardiac Angiography—Disneyland Contemporary Hotel, Orlando, Fla.
May 14-17	American Geriatrics Society—Hyatt Regency, New Orleans
May 14-17	Association of Clinical Scientists—Charleston Place Hotel, Charleston, S.C.
May 14-17	Virginia Society of Ophthalmology, annual meeting—Sheraton Tysons Corner, Northern Virginia
May 17-20	American Ophthalmological Society—La Costa, Carisbad, San Diego
May 17-21	American Urological Association—Hilton, Anaheim, Calif.
May 19	American Medical Student Association—New Orleans
May 19-21	American Society for Artificial Internal Organs—Marriott Marquis, New York
May 23-27	American Fracture Association—Hyatt Regency, Chicago
May 24-28	American Association on Mental Deficiency—Hilton, Los Angeles
May 25-30	American Association of Suicidology—San Francisco
May 26-27	American Otological Society—Fairmont, Denver
May 26-30	American College Health Association—Chicago
May 27-30	American College of Sports Medicine—Las Vegas
June 1-5	International Conference on AIDS (sponsored by US Public Health Service and World Health Organization)—Washington Hilton, Washington, D.C.
June 7-9	American Diabetes Association—Hyatt Regency, Indianapolis
June 10-12	Endocrine Society—Indianapolis
June 11-14	American Association of Neuropathologists—Seattle
June 13-16	Eye Bank Association of America—Grand Hotel, Mackinac Island, Mich.
June 15-17	NIH Consensus Development Conference on the Management of Clinically Localized Prostate Cancer (cosponsored by National Cancer Institute)—Bethesda, Md.
June 26-27	Association of Pakistani Physicians—Chicago
June 28-July 2	American Orthopedic Society for Sports Medicine—Orlando, Fla.

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Giant Esophageal Fibrolipoma:

Case Report and Review of the Literature

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CHARLES TUCKER, M.D.

Introduction

We have recently managed a patient with a giant fibrolipoma of the esophagus. Review of the literature revealed that this tumor is rarely reported, and that the nomenclature is quite inconsistent. We have not found reference to the appearance of these lesions on computerized tomography (CT).

Case Report

A 66-year-old man went to his family physician with a six-month history of progressive difficulty in swallowing solids; he had lost 15 lb and frequently regurgitated after meals. He denied any symptoms of aspiration. Physical examination was within normal limits. Chest film (Fig. 1) showed massive mediastinal widening and an air fluid level, and barium swallow (Fig. 2) revealed a huge intramural mass extending from the thoracic inlet to the level of the pulmonary hilum. CT demonstrated that the mass was fatty and quite extensive (Fig. 3). Again the location was interpreted as intramural.

Flexible esophagoscopy was performed. The mass was believed to be intramural, and biopsy showed prominent lymphofollicular proliferation and interstitial changes consistent with an inflammatory polyp. We explored the patient through a right thoracotomy with a preoperative diagnosis of giant intramural lipoma. There was a large (14 × 5 cm), lobulated esophageal mass, which to our surprise proved to be intraluminal and pedunculated, attached to the cervical esophagus by a narrow pedicle (Fig. 4). Histologic examination showed a benign polyp with a myxoid stroma which was classified as a fibrolipoma. The patient had an uncomplicated postoperative course and was discharged on the ninth postoperative day. He has remained asymptomatic since his operation.

Comment

This case was unusual on several counts, including the rather spectacular size of the lesion, the paucity of recent surgical or medical litera-

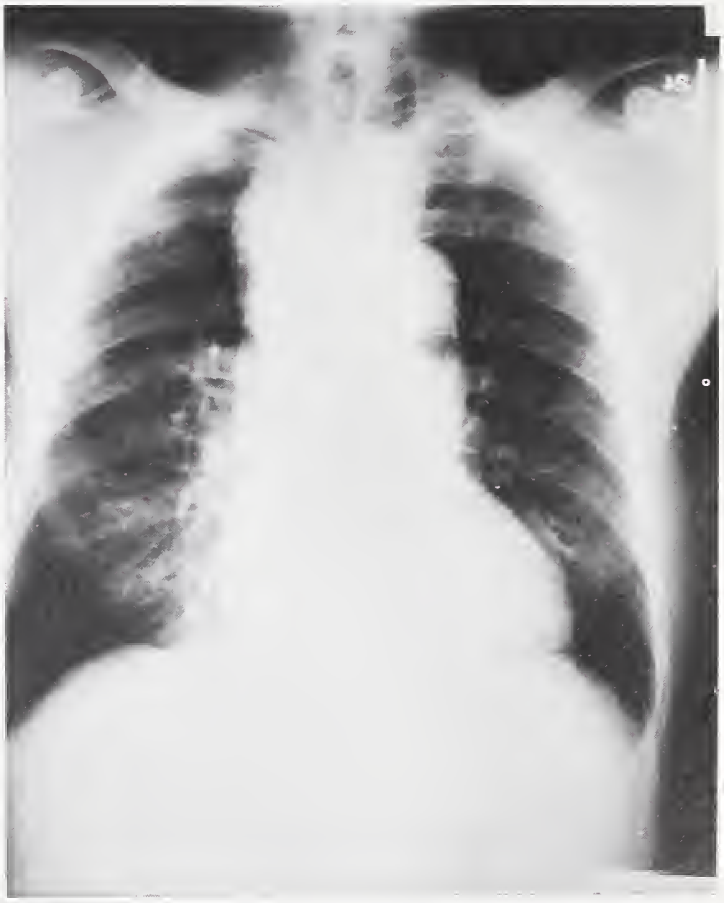

Figure 1. PA chest film revealing massive mediastinal widening and an air fluid level in the proximal esophagus just above the left clavicular head.

From the University of Tennessee College of Medicine, Memphis and Methodist Hospitals of Memphis.

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ture, and the use of CT in evaluation, which has not been previously described.

Benign esophageal tumors comprise about 20% of esophageal neoplasms.[1] Plachta[2] reported a series of 99 benign esophageal tumors in 1962, and found a total to that date of 432 cases in the literature. Schmidt and associates[3] reviewed the Mayo Clinic series, and divided these lesions based on location into pedunculated intraluminal or intramural lesions.

Pedunculated intramural tumors are unusual lesions. They were described prior to the development of roentgenographic techniques because of oral regurgitation of the mass. In 1922, Vinson[4] reported removal of an intramural pedunculated esophageal tumor via a transcervical route. This was the first such report in the American literature. In 1952, Bernatz and associates[5] reported a series of six surgically treated cases and noted the rather bewildering nomenclature of these lesions. Since these tumors are generally made up of mesenchymal elements and are described by the predominant pathologic appearance, they have been termed lipomas, pedunculated lipomas, fibrolipomas, fibrovascular polyps, myxomas, and various other combinations of these terms. They all tend to arise in the proximal esophagus, to be molded by the peristaltic action of swallowing, to have intact mucosal covering, and to be relatively asymptomatic in view of their size.

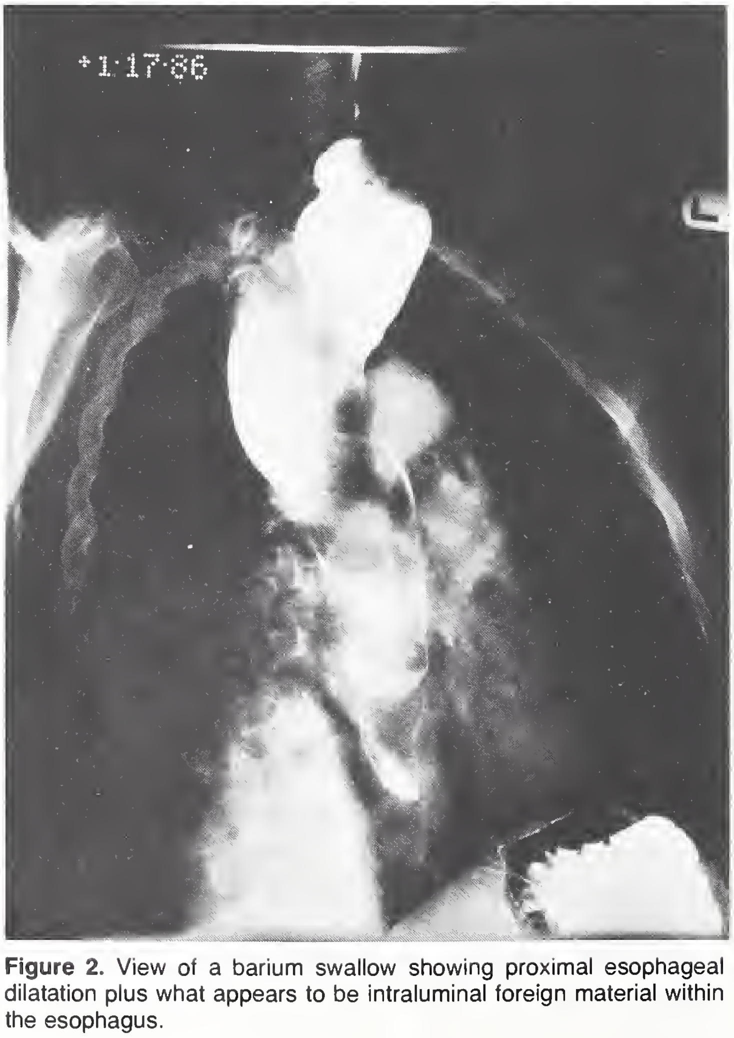

Figure 2. View of a barium swallow showing proximal esophageal dilatation plus what appears to be intraluminal foreign material within the esophagus.

This particular tumor was classified as a fibrolipoma. We found four case reports with most of the recent ones being in the radiology literature. Jang and associates[6] termed these tumors fibrovascular polyps, and stated that they were the most frequent intraluminal tumors of the esophagus. They reported three lesions, two removed via thoracotomy and a third removed transorally after regurgitation of the lesion. Lolley et al[7] reported the case of a patient with a long history of intermittent regurgitation of a large tumor. This lesion was managed via a transcervical route. Barki and colleagues[8] reported a similar case, resected via a cervical approach, and pointed out that malignant degeneration had been described. Lastly, Carter and Kulkarni,[9] who reported cervical excision of a similar large tumor, stated that 60 cases had been reported up to 1984, and recommended excision because of the potential danger of bleeding.

The use of CT has not previously been described in this setting. In this patient, the large fatty lesion was well delineated by its density and measured; the study was of considerable value in selecting a surgical approach. We did not correctly diagnose the polypoid nature of the lesion with endoscopy, barium swallow, or CT; this difficulty has been described by others and is due to the mucosal covering and huge size of the le-

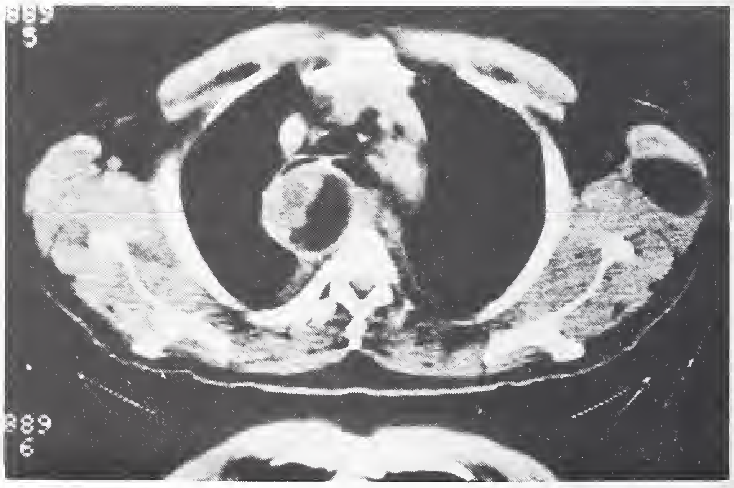

Figure 3. Computerized tomogram showing a fatty density which appeared to be in an intramural position.

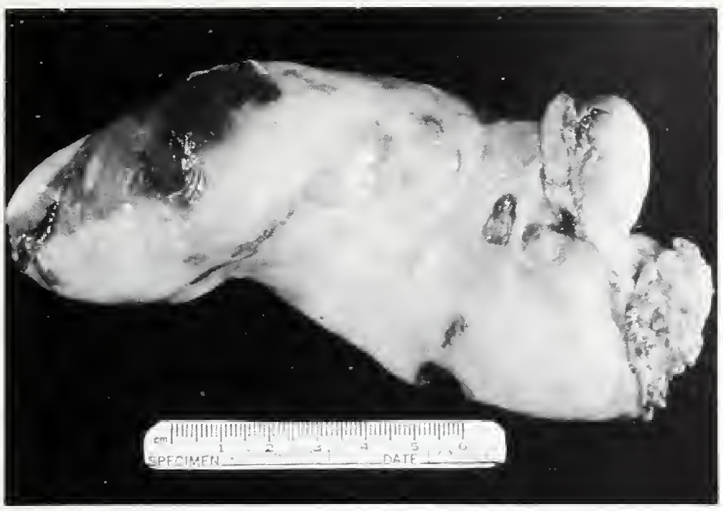

Figure 4. View of the large pedunculated tumor mass after excision.

sions. The major diagnostic confusion is with achalasia; the appearance of a large amount of radiolucent foreign material mixed with contrast but without obstruction of flow of contrast material at the gastroesophageal junction is the distinguishing feature.

Summary and Conclusions

Intraluminal pedunculated benign tumors are characterized by a bewildering nomenclature that at this point is probably uncorrectable. They should be excised when diagnosed because of progressive symptomatology, especially with weight loss, the propensity for regurgitation with fatal asphyxia,[10,11] the possibility of hemorrhage, and the remote chance of malignant degeneration. The frequent presence of large vessels in the stalk probably makes a direct surgical approach most prudent, although endoscopic removal has been achieved.

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Lymphangioma of the Spermatic Cord

PAIO FU HUANG, M.D. and JAMES B. FARNUM, M.D.

Unusual operative findings of an inguinoscrotal mass prompted suspicion of neoplasia, in spite of previous clinical differential diagnosis of spermatocele, hydrocele, and inguinal hernia; a frozen section diagnosis of lymphangioma was made. We present this experience with the clinical, gross, and microscopic material of a rare spermatic cord lymphangioma to facilitate sound decision-making in similar situations.

Case Report

A 57-year-old white male carpenter gave a history of recent treatment of reactive depression, emphysema, and hiatal hernia. An incidental finding was a right inguinal mass of two to three years' duration which caused intermittent, minor discomfort. There was no history of alcohol or tobacco abuse.

On physical examination, auscultation of the lungs revealed hyperresonance, decreased breath sounds, and diffuse wheezes bilaterally. There was a dime-sized, nontender, multinodular, mobile mass within the right spermatic cord, with possible extension to the epididymis. The overlying scrotal skin was freely movable over the mass. Laboratory data, including urinalysis, were unremarkable. An intravenous pyelogram showed normal kidney contours and bladder outline. The clinical impressions preoperatively favored spermatocele, hydrocele, and inguinal hernia, in order of decreasing probability.

Through a right transverse inguinal incision, a right orchiectomy with high ligation of the right spermatic cord was done. Dissection down through the external oblique aponeurosis revealed a 2.5-cm, soft, multinodular cystic mass which was several centimeters distal to the external inguinal ring. Frozen section diagnosis of a wedge biopsy was reported as lymphangioma. Further dissection freed the entire mass and testicle without difficulty.

The gross pathology specimen consisted of the right testicle, epididymis, and spermatic cord (Fig. 1). Fascial layers and cremaster muscle were dissected from the spermatic cord, revealing a tumor between the vas deferens and pampiniform plexus. The normal appearing testis measured 4.5 × 3 × 2.5 cm, and had an attached 5-cm length of epididymis. The vas deferens measured 12 cm from epididymis to resection margin and was paralleled by a 10-cm length of pampiniform plexus with a 15 × 7-mm transverse diameter. A soft, translucent, ovoid mass measuring 4.5 × 2.5 × 1.5 cm (Fig. 2), situated 2.5 cm from the epididymis and 3 cm from the resection margin, appeared well-defined, with pushing borders nestled between the vas deferens and pampiniform plexus. The mass did not appear to infiltrate adjacent structures. Transverse section showed a well-demarcated, multilocular cyst with internal cavities measuring .3 to .7 cm; they had thin fibrous walls and were filled with a clear, yellowish, mucoid

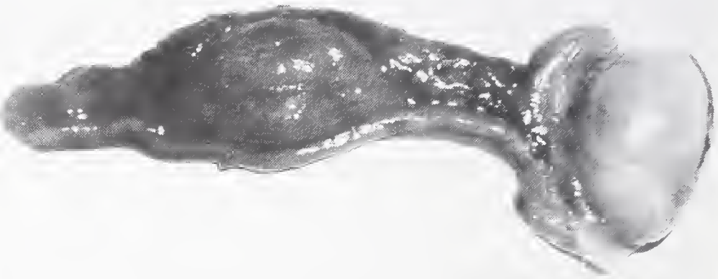

Figure 1. The surgical specimen shows a well-defined, ovoid tumor between the vas deferens and pampiniform plexus of the spermatic cord, with adjacent normal testis and epididymis.

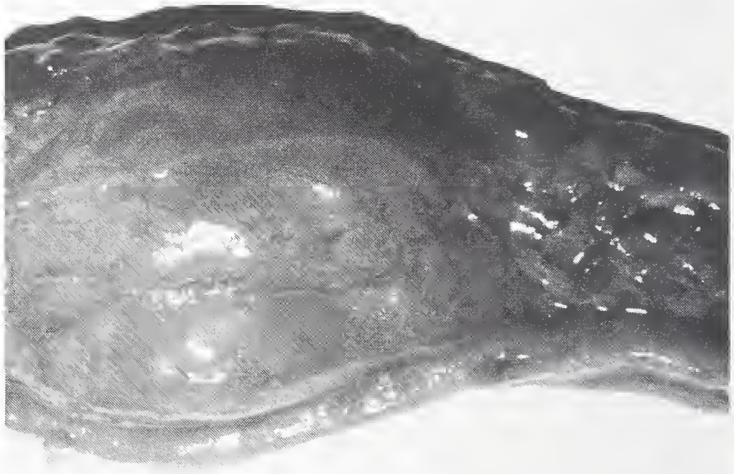

Figure 2. Close-up photograph shows a well-defined, translucent, ovoid tumor (4.5 × 2.5 × 1.5 cm) pushing between structures of the spermatic cord.

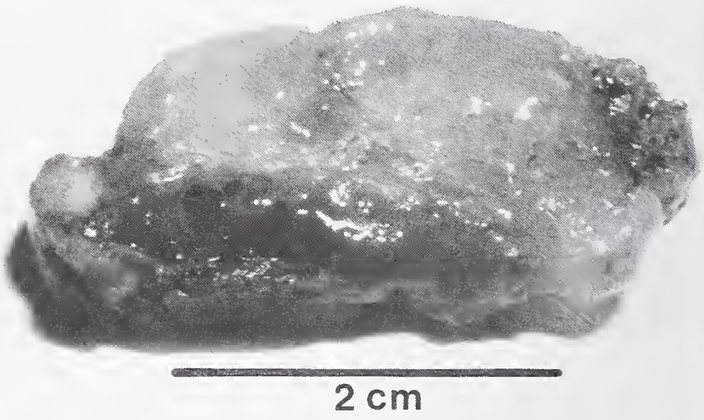

Figure 3. Transverse midsection of tumor reveals a multilocular cyst with irregular internal cavities filled with mucoid material and having thin fibrous walls. The vas deferens and pampiniform plexus are pushed to the extreme left and right edges, respectively.

From the Laboratory Service, Veterans Administration Medical Center, and the Department of Pathology, Quillen-Dishner College of Medicine, East Tennessee State University, Johnson City.

Reprint requests to Laboratory Service (113), Veterans Administration Medical Center, Mountain Home, TN 37684 (Dr. Farnum).

material that was easily extruded (Fig. 3). The testis, epididymis, and portions of remaining spermatic cord were normal.

Microscopic sections showed a neoplasm composed of irregular cystic spaces and of fibrous septa of variable thicknesses (Fig. 4). The cystic spaces were filled with a layered coagulum of proteinaceous material containing occasional clusters of lymphocytes; there was minimal extravasation into normal adjacent interstitium. Vascularization of the septal walls was moderate. Although the mass was independent of the vas deferens and pampiniform plexus, the tumor was pushing into the adjacent soft tissues (Fig. 5). The spaces were lined by endothelial cells, which in some areas were hyperplastic. The histological diagnosis was cavernous lymphangioma.

Comments

Lymphangioma, a benign neoplasm of the lymphatic system, is an unusual tumor found primarily in the young. Though it can arise anywhere in the body, it is rare in the inguinoscrotal location,[1,2] and is even rarer in the spermatic cord. Gueukdjian's excellent 1955 review of the world literature yielded 56 cases of lymphangioma of the groin and scrotum, of which only nine were specific to the spermatic cord[3]; these nine showed age variation from newborn to older adult.

The pathogenesis of lymphangioma is still uncertain. The most widely accepted theory, proposed by Goetsch,[4] is that these tumors arise from sequestration or failure of the lymphatic system to make connection with the venous system. Primitive lymphatic tissue has growth potential via sprouting, penetration, and secretion. The endothelial sprouts penetrate the adjoining tissue, the endothelial cells form droplets of secretion, and cyst-like spaces result from dilatation by secretions. Consequently, the adjacent tissue becomes compressed and fibrotic. This theory of origin would explain the pathologic and clinical features of lymphangioma.

Wegener's classification of simple (capillary), cavernous, and cystic lymphangioma is generally accepted[5]; combinations of the above patterns are frequently seen. Capillary (simple) lymphangioma, a congenital lesion of skin or mucous membrane of the head and neck area, is a slow-growing tumor composed of a network of endothelial-lined, thin-walled lymphatic spaces which are often separated by lymphoid aggregates. Lack of erythrocytes distinguishes the spaces from venules.

Cavernous lymphangioma is also a congenital lesion that grows slowly and is more common than the capillary type. It is composed of numerous dilated endothelial-lined spaces separated by a scanty connective tissue stroma. The margins of the tumor are not discrete. This type of lymphangioma occurs most commonly in the neck and axilla, and occasionally in the retroperitoneal area. The distinction from capillary lymphangioma on the basis of the size of the spaces is somewhat arbitrary; mixed cavernous lymphangioma and hemangioma are also common.

Cystic lymphangioma occurs principally in the neck. It is usually multilocular and contains serous fluid. The histologic features are similar to those of a cavernous hemangioma except for the size of the spaces. According to Keiller,[6] cystic lymphangioma is an advanced stage of the cavernous type resulting from changes in the drainage of the mass or alterations in the endothelial lining; Watson and McCarthy,[7] on the other hand, regard cystic lymphangioma as a distinct entity.

The differential diagnosis by physical examination in this case study included three entities

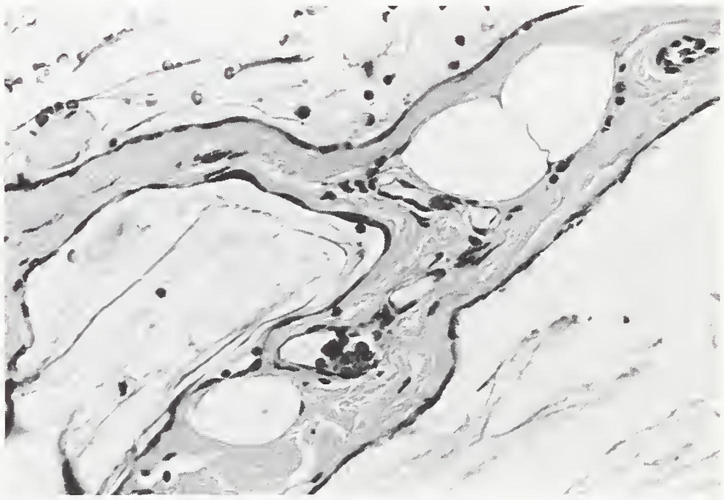

Figure 4. High power photomicrograph of the lymphangioma shows the edges of irregular cystic spaces and variable thickness of fibrotic septa. Note the flat endothelial cells lining the septae and the rich septal vascularization (hematoxylin-eosin, X400).

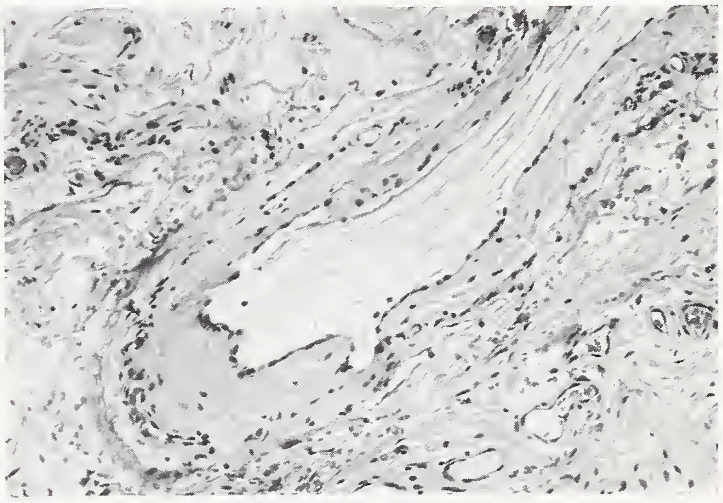

Figure 5. The margin of the lymphangioma shows an interdigitating, pushing, finger-like extension into adjacent soft tissues. This may account for the tumor's well-known propensity for recurrence (hematoxylin-eosin, X200).

frequently confused with inguinoscrotal lymphangioma, which may present as "scrotal surprises": inguinal hernia, spermatocele, and hydrocele.[8] Its high inguinal location suggested hernia, but due to its high location the examiner could feel only its cross-sectional tip; since its major extent was within or hidden by the inguinal ring, the size of the mass was underestimated. The proximity of the mass to the epididymis gave the impression of spermatocele or hydrocele, and its sharp demarcation of the mass further confused the clinical distinctions among hernia, spermatocele, and hydrocele. The slow growth of the mass matched the growth behavior of lymphangiomas, but its location may have precluded early notice.

Though surgical excision, external irradiation, sclerosing injections, fulguration, and cryotherapy have all been used in the treatment of lymphangioma, complete excision offers the best chance of cure. Recurrence is unfortunately frequent, and its incidence may be associated with increasing age. Those patients cited by Gueukdjian[3] with spermatic cord lymphangioma were treated by surgical excision with apparent absence of morbidity. In our case, orchiectomy with high ligation of the spermatic cord was curative, with no consequent complications in a follow-up period of three years.

Summary

We have presented a rare case of lymphangioma arising within the spermatic cord of a 57-year-old man, giving salient features of the gross and microscopic pathology, tumor pathogenesis and histology subtypes, clinical differential diagnosis and treatment options.

Acknowledgements

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Results of Cardiac Transplantation At St. Thomas Hospital

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SISSY DENTON, R.N., B.S.N.; BETH MOBLEY, A.C.S.W.; WILLIAM C. ALFORD, JR., M.D.;
GEORGE R. BURRUS, M.D.; WILLIAM S. STONEY, M.D.; CLARENCE S. THOMAS, JR., M.D.;
HENRY S. JENNINGS, III, M.D.; and EDWARD C. McDONALD, M.D.

Introduction

Human cardiac allografting began in December 1967[1] and was followed by a brief period when many centers performed heart transplants, an activity rapidly curtailed by poor results; only a small number of centers persisted. Since the introduction of cyclosporine (Cy) into the field of clinical transplantation,[2] in 1980, there has been a worldwide increase in the number of centers performing heart transplants. The Tennessee Heart Institute at St. Thomas Hospital instituted its program in March 1985, and since that time 42 patients have been referred for cardiac transplantation. Fifteen of these patients were considered candidates for orthotopic heart transplant (OHT).

Materials and Methods

Of the 15 candidates, 11 received orthotopic transplants, one died awaiting transplantation, one transferred to another transplant program, and two are presently awaiting an appropriate donor heart.

Of the 11 transplant recipients, there are ten men and one woman; their ages range from 30 to 54 years. All recipients satisfied the criteria in Table 1; they were compliant and well motivated, and had a strong support system. All had NYHA class IV symptoms and an estimated life expectancy of less than one year. None was a candidate for alternative medical or surgical therapy, and other contraindications to OHT, i.e., severe peripheral vascular disease, active infectious process, recent pulmonary infarction, irreversible renal and liver dysfunction, cancer, and elevated pulmonary vascular resistance (PVR), were excluded. The indications were idiopathic cardiomyopathy in seven patients, ischemic cardiomyopathy from coronary artery disease in three patients, and postpartum cardiomyopathy in one patient.

Donor selection criteria included age (men under 35 or women under 40), hemodynamic stability without the need for high dose inotropic support, and no history of prolonged cardiac arrest or previous cardiac disease. The particular combination of donor and recipient criteria was based on an approximate size and weight, ABO blood group compatibility, and lack of donor-specific antibodies. A negative prospective lymphocyte crossmatch was required if the recipient had evidence of preformed antibodies. Human leukocyte antigen (HLA) typing was performed only retrospectively. The average waiting time was six weeks. Three donor hearts were procured locally and the remaining hearts elsewhere; the greatest procurement distance was 576 miles. Total ischemic time ranged from 83 minutes to 192 minutes, with an average of 120 min-

TABLE 1

RECIPIENT CRITERIA FOR ORTHOTOPIC HEART TRANSPLANT

End-stage disease—NYHA class IV
Psychologically stable
No malignancy
No recent pulmonary infarction
PVR <6 Woods units*
No systemic illness
No active infection

*Pulmonary vascular resistance.

From the Tennessee Heart Institute, St. Thomas Hospital, Nashville.

Reprint requests to 4230 Harding Road, Suite 501, Nashville, TN 37205 (Dr. Lea).

utes. The operative technique was that of Lower and Shumway[3] consisting of a left atrial anastomosis, followed by a right atrial anastomosis, and then anastomosis of the heart to the aorta and pulmonary artery.

Postoperatively, the recipients were placed in a reverse isolation environment during the first week. Mask isolation was continued until discharge from the hospital. Postoperative care was quite similar to the care of the more conventional cardiac surgical patient. Vital signs, intake and output, right heart pressure data, and cardiac output were monitored for 24 to 48 hours. Mechanical ventilation was discontinued within the first 24 hours. In the second posttransplant week, a cardiac rehabilitation exercise program was started and the recipient received instructions regarding immunosuppression and his medications. After transplantation, patients have met monthly with the transplant social worker in a support group which discusses common problems and goals.

Immunosuppressive therapy consisted of Cy, rabbit antithymocyte globulin (RATG), azathioprine (AZA), and corticosteroids. Patient rejection surveillance was based primarily on endomyocardial biopsy (EMB).

Immunosuppressive Regimen. Currently, the recipient receives preoperative AZA (1 mg/kg) and an initial oral loading dose of Cy (12 to 15 mg/kg) based on renal function. Methylprednisolone (1 gm IV) is administered on induction of anesthesia and again after termination of cardiopulmonary bypass. In addition, patients receive RATG (0.2 mg/kg IV) at termination of cardiopulmonary bypass and daily for five to seven days. Methylprednisolone (125 mg IV) is continued every eight hours postoperatively for three doses. AZA (1.0 to 1.5 mg/kg) is continued twice daily beginning on the first postoperative day. Beginning on the second postoperative day, methylprednisolone (100 mg IV) is administered twice daily and reduced by 10 mg every other dose. When the patient can take oral medication, prednisone replaces the methylprednisolone in equivalent dosages. Beginning on postoperative day 10, prednisone is reduced by 1 mg every other dose until a maintenance of 20 mg/day is reached at three weeks. Cy therapy is begun on the second postoperative day with a dosage of 7 mg/kg. Subsequent Cy dosage is adjusted to maintain Cy through serum levels between 250 and 350 ng/ml using high pressure liquid chromotography (HPLC) assay and normal blood urea nitrogen and serum creatinine levels.

Beginning at three months, the Cy dosage is reduced to maintain serum Cy levels between 150 and 250 ng/ml, with an average daily dose of 2 to 4 mg/kg. Also beginning at three months, prednisone is tapered to a dosage of 5 mg/day. The reduction in Cy and prednisone dosage is made in conjunction with the results of the EMB.

AZA is adjusted to maintain a leukocyte count greater than 4,000/cu mm and a platelet count greater than 100,000/cu mm.

Daily "pulses" of methylprednisolone (500 to 1,000 mg IV) are given for three days to treat rejection. For severe rejection or if rejection persists, further pulses of methylprednisolone are administered with the addition of RATG.

Immunologic monitoring consists of absolute lymphocyte counts, lymphocyte morphology as prepared by buffy coat preparations, T-lymphocyte subgroups numeration, and EMB. Treatment of rejection is based solely on the EMB.

EMB, using the technique described by Caves and co-workers,[4,5] is performed frequently during the first three months, monthly up to six months, and then at six- to eight-week intervals. At 12 months, patients undergo left heart catheterization, coronary angiography, and right ventricular biopsy with subsequent EMB performed at three-month intervals. Small fragments of right ventricular endocardium are stained by hematoxylin

TABLE 2

COMPLICATIONS

Rejection	12
Major infection	8
Minor infection	15
Hypertension	9
Transient psychosis	6
Leukopenia (wbc <1,500)	5
Arrhythmias	
Atrial	3
Ventricular	1
Seizure	1
Diabetes	1
Hyperkalemia	1
Peptic ulcer disease	1
Pneumothorax	
Spontaneous	2
Iatrogenic	1
? Lymphoma	1

and eosin, trichrome, and methylpyronine. The diagnosis of rejection is according to the criteria of Billingham.[6]

Results

Of the 11 patients receiving transplants, eight are alive and well and living at home, having survived between 8 and 20 months; three have died, one from donor heart failure at time of transplantation, one from accelerated acute rejection in the early postoperative period, and one from acute rejection in the tenth month posttransplant. In both patients who died of acute rejection, treatment for rejection was instituted but was unsuccessful. These patients rapidly deteriorated and died within 24 hours. In the eight remaining patients, there have been five episodes of rejection in four of the patients. These have responded well to rejection therapy.

Complications have been related primarily to allograft rejection and immunosuppression (Table 2). The complications have occurred primarily in the early posttransplant period, 85% of them in the first six months. The majority (52%) of the complications were either rejection or infection. The infections are summarized in Table 3. Over half of the eight major infections were pulmonary related. Nine of the 15 minor infections were mild to moderate episodes of gastroenteritis lasting from 24 hours to four to five days. All patients have developed Cy-related diastolic hypertension at three to four months posttransplant. This has been managed successfully by decreasing Cy dosage and administering afterload reducing agents and calcium channel antagonists.

The quality of life of the OHT patients has continued to improve. All surviving patients have returned to a near-normal lifestyle; five patients have undergone recatheterization after one year. End diastolic volume, end systolic volume, and subsequent ejection fraction (EF) calculation were derived using a validated computer program for left ventricular volume measurement based on Simpson's Rule. All have demonstrated good left ventricular performances without regional wall abnormalities. EF have ranged between 56% and 76%. No significant coronary artery disease has been demonstrated.

TABLE 3

INFECTIONS

Major	
Pneumocystis pneumonia	3
Tuberculosis	1
Pneumonia (unspecified)	1
Herpes zoster	1
Urinary tract	1
Cytomegalovirus + serology	1
Minor	
Gastroenteritis	9
Otitis	1
Sinusitis	1
Herpes simplex	3
Wound infection	1

The eight surviving OHT patients have a cumulative follow-up of 108 patient-months. The duration of survival ranges from 8 to 20 months. The actuarial one-year survival,[7] excluding perioperative mortality, is 83.4%. This compares favorably with the June 1986 report of the registry of the International Society for Heart Transplantation,[8] which showed an 87.14% one-year survival for similar patients.

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Special Item

Social Security Disability—
A Greater Role for Treating Physicians

DAVID A. ETTINGER

Following a recent federal court order in a class action lawsuit, the Social Security Administration (SSA) will place increased reliance on treating physicians, rather than hired consultants, in determining eligibility for disability benefits. In *Samuels v. Bowen,* No. 82-2827 (W.D. Tenn. 8/20/86), the court ordered SSA to adopt the following procedures: (1) SSA must ask treating physicians to evaluate a patient's ability to sit, stand, walk, and lift; (2) when there is a conflict between treating and consulting physicians, SSA must give greater weight to the opinions of treating physicians regarding the severity of an impairment and resulting limitations, as long as the treating physician is qualified and submits supporting clinical or laboratory findings; and (3) when additional tests or examinations are needed, SSA must attempt to purchase those services from a treating physician before sending the patient to a consultant. As a result of these procedures, physicians are now able to strongly influence disability determinations and improve the quality of those determinations.

SSA has two standards for determining disability. First, any patient who has an impairment described in the Social Security Listing of Impairments or impairments equal in severity to an analogous described impairment will be found disabled. The Listing of Impairments is a document written by and for physicians. Copies can be obtained from Professional Relations, Disability Determination Section, P.O. Box 775, Nashville, TN 37202. The decision that a listed impairment is present or equaled is a purely medical decision, and does not take into consideration vocational factors. It is important to remember that many patients will not have a listed impairment, but may still qualify for disability benefits.

The *Samuels* decision offers physicians the opportunity to help establish the presence of a listed impairment in two ways. First, many of the listings involve subjective judgment. Several of the cardiac listings, for example, require the presence of angina. In the past, consulting physicians have frequently concluded, based on a single examination or a review of records with no examination, that a patient's chest pain is not "typical" of angina. In the future, the treating physician's opinion, consistent with medical findings, should be readily accepted by SSA. Second, many listings depend on specific test results. The respiratory listings often depend upon pulmonary function studies. Results obtained by hired consultants are frequently invalid because the patient failed to understand instructions or expend adequate effort. It is likely that these problems will be significantly reduced when the testing is performed by a treating physician.

The second disability standard depends upon the patient's age, education, work history, and, most importantly, remaining capacity to work. A patient will be found disabled if, as a result of an impairment, he can no longer perform past work and cannot, after considering vocational factors, learn a significant number of less demanding jobs within his remaining capacity. For example, a 55-year-old patient with a high school education and past work as a laborer will be found disabled if he cannot lift 25 pounds frequently and up to 50 pounds occasionally.

Under the *Samuels* order, SSA will now ask treating physicians to assess a patient's remaining ability to perform work functions. (SSA will send

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an assessment form that calls for a time-consuming narrative response. I will provide a simple check off form to physicians upon request.) That assessment, if supported by clinical or laboratory findings, should be accepted by SSA. In many cases, the determination of disability will depend exclusively on whether the patient can stand or walk six hours during an eight-hour day or on the maximum weight a patient can lift or carry occasionally and frequently. These assessments are inherently difficult. The court order recognizes that a treating physician is better able to make an accurate assessment than a consultant who has never examined the patient or has performed just a single examination.

The effect of the *Samuels* order on the quality of disability determinations will depend largely on the response of physicians. Many physicians have become discouraged with the disability process and provide minimal response to requests for information about their patients. A conclusory statement that a patient is "disabled" will continue to carry little weight and merely create false hopes for the patient. Physicians who decline to perform necessary examinations or tests at SSA expense will continue to have their patients evaluated by one-time consultants. On the other hand, physicians who submit detailed reports, based on treatment records or additional testing at SSA expense, establishing the severity of a patient's impairment and limitations should see a marked improvement in the quality of disability determinations. (Errors will still occur. Patients can be referred to the nearest legal services office for help with an appeal. The address and telephone number can be obtained by calling 615-242-0438.)

Not every patient who applies for disability benefits will meet the stringent disability standards. For those who do, a favorable decision without lengthy appeals can mean the difference between indigency and a subsistence income. An award of disability benefits may also result in eligibility for Medicare or Medicaid, increasing the patient's ability to complete a course of treatment.

The court order in *Samuels v. Bowen* offers physicians an opportunity and a challenge. The opportunity is to assist disabled patients to qualify for Social Security insurance benefits. The challenge is to provide high quality information that vindicates the court ordered preference for evidence from a treating physician.

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Siriasis

CHARLES E. KOSSMANN, M.D., Editor

STEVEN K. SOUTHER, M.D.

(Resident Physician)

A 64-year-old black woman was brought to the emergency room of THE MED after family members living in an adjoining apartment heard her fall. They found her lying on the floor, awake but disoriented, lethargic, and unable to talk. Her apartment was noted to be warm and humid but not excessively hot even though all windows were closed. In the five days before admission the atmospheric temperature had been unusually high for the time of the year (early June 1985), varying from a maximum of 95°F to 97°F to a minimum of 75°F to 77°F. There was no history of recent physical exertion or recent illnesses other than a mild productive cough for a few days. Although she had a history of regular, heavy ethanol use, she had not imbibed during the preceding 48 hours. Her only medication was an antitussive (Robitussin). The recent cough, productive of whitish sputum, was not accompanied by fever, chills, or chest pain. An occipital headache associated with a feeling of profound fatigue and light-headedness had begun one day before admission.

There was no history of previous chronic illness or admission to a hospital. A 20-year history of ethanol use consisted primarily of "binge" drinking of 1 to 2 pints of whiskey per week; she had smoked a pack of cigarettes daily for 30 years.

Her rectal temperature was 107.2°F (41.7°C), heart rate 136/min and regular, respiratory rate 32/min and regular, blood pressure 140/40 mm Hg in both arms, and unchanged with body position. She was morbidly obese, lethargic but easily arousable, and oriented only to person and place. The skin was extremely hot and dry but with good turgor. There were no purpuric or petechial hemorrhages. The pupils were equal, round and reactive to light. There was no episcleritis but several small conjunctival hemorrhages were present bilaterally; the fundi were benign. Mucous membranes of the mouth and throat were moist. The jugular veins were not distended. Some atelectatic pulmonary rales were cleared partially with cough. Except for a hyperdynamic apex and rapid rate, the heart was negative. Peripheral pulses were bounding. The bowel sounds were hypoactive. Examination of the genitourinary tract, rectum, and extremities was unremarkable. Neurologically, there was general lethargy, mild confusion, and mild generalized motor weakness. Deep tendon reflexes were slightly but symmetrically depressed in both the upper and lower extremities. The cranial nerves, sensory sensation, and cerebellar function were normal; there was no Babinski sign.

The hematocrit was 39.7% with a mean corpuscular volume of 97.7 cu μm/cell. The phase platelet count was 152,000/cu mm. The white blood cell count was 6,500/cu mm, with 1% band forms, 70% segmented neutrophils, and 25% lymphocytes; there were 4% monocytes. The urine was abnormal only for the presence of 2+ bilirubin. Electrolytes in mEq/L were: sodium 138, potassium 4, chloride 101, and bicarbonate 22. The serum BUN was 19 mg/dl, creatinine 2.5 mg/dl, and glucose 169 mg/dl. The thoracic roentgenogram revealed a normal cardiac silhouette and left basilar atelectasis. Except for sinus tachycardia the electrocardiogram was normal. Arterial blood gases on room air corrected for temperature showed a pH of 7.45, $P\text{CO}_2$ of 25 mm Hg, $P\text{O}_2$ of 62 mm Hg (86% saturation), and a bicarbonate of 17 mEq/L.

The patient was initially treated in the emergency room with cold water sponging and application of ice packs to the groin and both axillae. This resulted in reduction of the rectal temperature to 101.3°F (38.5°C) within 90 minutes. Then, following transfer to the intensive care unit, she became transiently hypotensive with a systolic blood pressure of approximately 80 mm Hg. This responded well to crystalloid infusion, and did not recur.

Serial liver function tests showed an increase in the serum SGOT from 63 to 162 U/L, the SGPT from 36 to 64 U/L, and the LDH from 259 to 361 U/L. Although mild diffuse muscle tenderness and a rise in the serum CK to 1,335 U/L occurred, the urine myoglobin was found to be negative on two successive determinations. The CK MB fraction rose to a maximum of 11.4 U/L at a time when the total CK was 1,335 U/L but serial electrocardiograms remained unchanged except for resolution of the initial tachycardia. A diffuse intravascular coagulation (DIC) profile showed a mild prolongation of the prothrombin time to 13.4 seconds, fibrin-split products greater than 40 mEq/dl, and a fibrinogen of 203 mg/dl (normal 200 to 400 mg/dl) with a slight decline in the platelet count to 135,000/cu mm. Protamine sulfate determination was negative and the peripheral smear showed no evidence of hemolysis.

The hospital course was complicated by the development of a left lower lobe pneumonia which was treated successfully with intravenous antibiotics. Following resolution of the pneumonia the patient was discharged home two weeks after admission in good condition.

Final Diagnosis: Heat stroke; left lower lobe pneumonia.

MICHAEL L. ISAACSON, M.D.

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In the nebula Orion can be found the star *Sirius,* the brightest in the heavens, located in the constellation *Canis Major.* The word *Sirius,* from the Greek, means the scorcher. Hence "take heed of Caesar and his perspiring legions and beware of the season when *Sirius,* the Dog Star, rises shortly before the sun."[1] Thus from earliest bib-

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Presented July 31, 1985.

lical times comes the expression "the dog days of July and August," when *Sirius,* the dog star, arises with the sun.

Siriasis, a disease produced by the heat of the sun, is better known as sun stroke. Impressive throughout history has been the magnitude of heat-related deaths during particularly oppressive heat waves. One such episode occurred in Peking in July of 1743 when 11,000 people were reported to have perished.[2,3] While this number is indeed devastating, close to 4,000 heat-related deaths still occur every year in certain humid portions of the United States.[4,5] In terms of mortality, heat stroke is not a major problem compared to automobile accidents, heart disease, or cancer, but as a preventable disorder, it is a significant health problem with an unacceptably high yearly mortality rate.[6]

Pathophysiology

In discussing the pathophysiology of heat illness, it is important to recall that the body temperature is determined by three factors: environmental temperature, body production of heat, and dissipation of heat from the body. At 95°F (35°C) as much as 150 kcal/hr can be absorbed by the body from direct sunlight.[5,7] Heat production is the result of two factors: basal metabolism, ranging from 65 to 85 kcal/hr, and energy metabolism, or work, ranging from mild work at 300 kcal/hr to strenuous work at 900 kcal/hr.[7,8] The heat generated is dissipated by conduction—molecular transport of heat from the skin to the environment; convection—heat from the body core moved by blood to the skin where it is then reduced by cool air; radiation—heat transferred by electromagnetic waves to the surrounding environment; and, finally, evaporation—sweat vaporized to act as a coolant.[8] These multiple effects can by simplified by this equation[1]:

$$\text{Core body temperature} = \text{metabolism} \pm \text{radiation} \pm \text{conduction} \pm \text{convection} + \text{work} - \text{evaporation}$$

When ambient temperature exceeds body temperature, conduction, convection, and radiation no longer aid the body in dissipating heat. In fact, they can quite often contribute to the heat gain. Sweating is left as the only mechanism of heat dissipation and if the humidity is high, as it often is in Memphis, this mechanism can be impaired as well. Maximum sweating in an untrained individual accounts for approximately 650 kcal/hr loss of heat.[3] Such an individual exercising in a hot environment may not be able to keep up with the pace of heat production without gaining heat. As the core temperature increases, the hypothalamic thermoregulatory center is stimulated by the increased temperature of the perfusing blood and by sensory input from peripheral thermoreceptors. As a result, compensatory cooling mechanisms are initiated, shivering is inhibited, sweating occurs, cutaneous vasodilation occurs and local cord reflexes are activated.[2,9]

In discussing the pathophysiology of heat illness, it is necessary to include a discussion of acclimatization, preferably designated acclimation. Two basic physiologic responses are involved. One is decreased heat production, the other is increased heat dissipation. A significant level of acclimation can be acquired in 10 to 20 days with only short daily periods of exposure to the heat. However, more often the process takes as long as two months.[10]

There are five basic adaptations that occur during acclimation. Metabolic efficiency is enhanced. Skeletal muscle glycogen stores increase as do the number of mitochondria in cardiac and skeletal muscle. This is important because more aerobic than anaerobic metabolism is occurring. The former is approximately 20 times more efficient, producing more ATP with less heat. Sweating is initiated at a lower level of core temperature with an increased rate of sweating from approximately 1.5 L/hr in an untrained individual to as high as 2.5 to 3.0 L/hr in an acclimated person. Myocardial efficiency is improved, maximum cardiac output and stroke volume are increased, and peak heart rate is decreased. Conservation of sodium by the kidneys and sweat glands occurs through an increased secretion of aldosterone. In unacclimated individuals, there may be as much as 30 to 50 mEq of sodium lost per liter of sweat, whereas in acclimated individuals this drops to as little as 2 to 5 mEq/L. Plasma and extracellular fluid volumes increase, thus altering cutaneous circulation and heat dissipation ascribable to three factors: increased activity of the renin-angiotensin-aldosterone system, increased sodium and water retention, and increased antidiuretic hormone production with expansion of body water, in some acclimated individuals by as much as 7%.

There is one deterimental effect of the acclimation process that can be instrumental in the development of heat stroke at times, namely loss of potassium. It occurs during the early phases of acclimation as a result of losses in the sweat and urine of potassium due to increased aldosterone secretion. An average deficit of 517 mEq of potassium has been recorded in untrained military recruits undergoing intense physical conditioning in hot areas. This trend, however, is usually corrected by the end of the acclimation period.[2,3,8]

Clinical Spectrum

There is a spectrum of heat illness that ranges from heat edema to heat stroke. The latter, with several subsets, is a major illness with a high mortality.

Heat edema consists of minimal swelling of the feet and ankles. It is ascribed to vasodilation of the cutaneous and muscular vessels with capillary leakage into the interstitium of the lower extremities. The treatment consists of skillful neglect with avoidance of overly vigorous diagnostic and therapeutic intervention. Simple elevation of the extremities and use of support hose will suffice, with subsidence of the edema as acclimation progresses.[10,11]

Heat syncope appears to be a variant of vasovagal syncope, and is initiated by arteriolar dilatation. The vagal release results in compensatory tachycardia. The syndrome can be made worse by the volume loss secondary to sweating. The treatment is to rule out other causes of syncope, with volume replacement if necessary. Prolonged standing must be avoided and prophylactic measures promptly instituted if prodromal symptoms appear.[10,11]

Heat cramps are acute muscle cramps. They are excruciating at times and quite often seen even in the well-trained, acclimated individual. This syndrome results from excessive loss of sweat, and even though sodium conservation mechanisms are operating, the higher volume of fluid loss results in depletion of the serum cations or even the total body stores. It is believed to be due to secondary involvement of the calcium-dependent relaxation mechanisms of skeletal muscle. Rest, a cool environment, salt replacement, and possibly massage are standard therapy. No significant muscle injury has been described as due to heat cramps alone. Table 1 lists some common replacement fluids used for prevention and treatment of heat cramps. The two on the bottom, Sportade and Gatorade, are the two that have the most sodium. Sportade, with a greater content of potassium, may be the best one in the early days of acclimation when there are high losses of that cation.[1,2,6,12-14]

TABLE 1*

SODIUM AND POTASSIUM CONTENT OF COMMON REPLACEMENT FLUIDS

Liquid	Sodium (mEq/L)	Potassium (mEq/L)	Calories (per 8 oz)
Cola	6	<1	110
Orange Juice	<1	43	108
Tea (plain)	1	1	2
Beer	1	4.7	114
Sportade	27	14-20	90
Gatorade	21	3	40

*From O'Donnell[13] with permission.

Heat exhaustion may be of two types: predominantly water depletion or predominantly salt depletion. The water depletion form occurs when the water supply is limited or unavailable. It can be seen in such diverse groups as soldiers or laborers in a hot environment and in infants and feeble adults who cannot express their desire for water. Symptoms are intense thirst, weakness, marked CNS dysfunction—athetoses, tetany, muscular incoordination and sometimes frank psychosis, which can make it difficult to differentiate from heat stroke.[3] The treatment is to move the patient to a cool environment, remove the clothing, and administer isotonic intravenous (IV) solution.[6,13]

The predominantly salt depletion type usually occurs in a different setting, similar to what is encountered in heat-stressed subjects before acclimation. There are large volumes of thermal sweat, with adequate intake of water but not of salt. The symptoms are similar to those encountered with heat stress in an unacclimated individual but with the addition of nausea, vomiting, diarrhea and skeletal muscle cramps as well. The patient looks sick, appears haggard and pallid, and displays clammy skin with poor turgor. The treatment is to remove him from the hot environment, remove all clothes, and give IV fluids. The fluid used is usually isotonic, but sometimes if the sodium depletion is extreme, it may be necessary to use hypertonic solutions such as 3% sodium chloride.[3]

Heat stroke by definition is characterized by two features—an elevation of the core temperature to greater than 105.8°F (41°C) and severe CNS dysfunction. Anhidrosis, the lack of sweating, may or may not be present.[6] In heat stroke the thermoregulatory center is overwhelmed by a devastating rapid rise in core temperature.

Subsequently the entire system falters.[1] The essential morbidity appears to be of two types—one is cellular damage by direct thermal injury, with denaturation and activation of enzymes, destruction of cellular membranes, and alteration of chemical bonds; the other is cellular hypoxia due not only to increased metabolic demands but also to circulatory insufficiency. It usually is not difficult to diagnose. However, since sweating may be present and sufficient cooling may have occurred before the patient is seen, the hyperpyrexia may have abated so that a high index of suspicion of the syndrome is required. Basically, any person who loses consciousness under conditions of heat stress is suspect.[6]

The differential diagnosis of heat stroke is wide and includes almost anything that can cause high fever. Of particular note are anticholinergic syndromes from toxic doses of drugs such as atropine, salicylates, phencyclidene (angel dust), and amphetamines.

Symptoms of heat stroke are many. An altered mental status is characteristic—agitation, psychosis, confusion, coma. Cerebellar dysfunction, ataxia, and dysarthria may predominate. Motor deficits are common—hemiparesis, quadriparesis and sometimes peripheral neuropathy. Other neurologic findings indicating CNS damage include fixed dilated pupils, and decerebrate or decorticate posturing. There can be anhidrosis, tachycardia, hyperventilation, hypotension, and sometimes jaundice.[6] It has been demonstrated that loss of consciousness can occur with few other symptoms in 71% of patients with heat stroke.[15]

Classes of Heat Stroke: There are two types of heat stroke, *classic* or *sedentary,* and *exertional.* The classic form typically occurs in epidemics during heat waves and affects primarily the elderly and infirm. In most series, the average age is 70 years and older. Most of the patients have underlying chronic disorders such as heart failure, diabetes mellitus, or chronic alcoholism, and many are taking medications such as diuretics, anticholinergics, or phenothiazines.[12] These epidemics are likely to occur in large cities, which include clusters of urban dwellings that have been appropriately termed, "heat islands."[16] One study from Kansas City during the heat wave in 1980 demonstrated that a rise in cases of heat stroke does not occur until about the fifth day of abnormally high temperatures (as in today's case); the peak prevalence is usually around the 14th day.[17] Discussing the cause is basically a matter of considering fluid loss, usually in the elderly, with a heart and circulatory system that cannot compensate for the fluid loss and the increased metabolic demand. The result is a rapid rise in temperature. Usually there is a prodromal period characterized by nausea, vomiting, muscle cramps, headache, weakness, and confusion.

Exertional heat stroke is usually seen in soldiers, athletes, and those who must make enormous metabolic demands on their muscles in a hot environment. The precipitating factor is rapid, strenuous muscular exercise. An often associated precipitant is hypokalemia.[12] Symptoms are as above, but sweating occurs in more than 50% of individuals with exertional heat stroke.[3]

With regard to laboratory manifestations, serum sodium is variable, potassium is low, and calcium, magnesium and phosphorus are usually low. On the other hand, hypercalcemia in certain individuals may occur in about the second week of the syndrome.[18] Glucose is variable, and the BUN usually goes up, as does the serum uric acid. There are marked serum enzyme elevations from the heart, liver, muscle and kidneys; quite often the serum amylase will go up as well. Usually there is a metabolic acidosis, but respiratory alkalosis is not uncommon. The hematocrit is variable; leukocyte counts can range from 20,000/cu mm to 50,000/cu mm. The coagulation system is greatly disturbed. Urinary casts, both red cell and white cell, and proteinuria are also seen.[6,11]

The arterial blood gases may be abnormal, but it is most important that the values reported be corrected for temperature. This should be done for anybody with fever but particularly for patients with the hyperthermia of heat stroke. For every one degree rise of temperature of the blood there is an increased consumption of O_2 and production of Co_2 by 6.0% and 4.4% respectively, while the pH will fall by about 0.147%.[19] Thus, a patient may initially have what appears to be a respiratory alkalosis with hypoxemia, but when a correction is made for temperature, there is actually a metabolic acidosis without hypoxemia.[20]

To expand on the potential role of potassium deficiency requires consideration of three factors. A depleted body potassium tends to render skeletal muscles more susceptible to injury. This is believed to be due to the fact that potassium is a potent vasodilator. Without this vasodilation in the face of a high oxygen demand, ischemia and necrosis may occur. Potassium deficiency impairs glycogenesis, a process necessary for heat-stressed muscle in an acclimated state. Hypokalemia can cause postural hypotension. The mechanism is not certain but is thought to be a relative unresponsiveness to the vasoconstrictive action of catecholamines.[3]

Physical Examination. All of the findings previously mentioned in heat exhaustion can be seen with heat stroke, to which is to be added decerebrate and decorticate posturing. As stated by Hamilton, "it does not take long either to boil an egg or to cook neurons."[21] Even with manifestations of cerebral dysfunction many patients will recover completely. Seizures are common. In one series of 158 people, 60 demonstrated focal or generalized seizure activity.[22] The spinal fluid, though usually normal, can contain blood and sometimes an increase in protein. The pathologic causes of the clinical findings are edema of the brain and leptomeninges, with punctate hemorrhages, degeneration of cells, chromatolysis, and swollen dendrites. The Purkinje cells may show pyknotic nuclei especially in the cerebellum.[15]

Cardiovascular Aspects of Heat Stroke: Survival of the patient with heat stroke depends upon an adequate response of the heart. The cardiac output may have to increase to 6 L/min/sq m in order to meet the body's elevated demands, particularly of the skin and muscles.[23] With exertion there is in addition the factor of exercise hyperemia. Muscular work can shunt as much as 4 L/min of blood to the muscles. There may also be a substantial loss of plasma volume into the muscle cells during this process. As the muscles' major fuel is glycogen, with glycolysis water moves from the plasma into the cells; as much as 10% of the arterial volume flowing to the muscle may become intracellular.[3] To summarize, the increased plasma flow to the muscles and skin with fluid loss through sweat and transfer into cells, even if partially counteracted by a decrease of hepatic flow by 15% to 60% and increased generalized splanchnic vasoconstriction, can cause a net deficit in plasma volume requiring a compensating increase in cardiac output of up to 125%. The clinical result is a hyperdynamic cardiovascular system with a bounding precordium and pulses and a wide pulse pressure. These features can persist for hours after the patient has been cooled back to normal.

High output cardiac failure or cardiogenic shock can occur as complications. Three mechanisms have been proposed to explain these potentialities: hemorrhage (see below) with resultant impaired myocardial function; myocardial degeneration due to direct thermal injury and myocardial hypoxia; elevation of pulmonary vascular resistance of unknown cause.[3,5,6,23] Gold[24] exposed normal human volunteers to extremes of heat and demonstrated that as heat storage rose and produced a hyperkinetic circulation, the mean venous pressure rose from 40 mm H_2O to as high as 200 mm H_2O. He postulated that circulatory collapse was the result of high output cardiac failure, and that sweating ceased due to the increased mean venous pressure.

Sometimes there are slight plasma elevations of the MB fraction of the CK enzyme without other evidence of cardiac damage. Electrocardiograms can show sinus tachycardia, nondiagnostic abnormalities of ST and T, and some prolongation of the QT interval.[11,24]

At necropsy there is right-sided cardiac dilatation, petechial and gross myocardial hemorrhage, degeneration and necrosis of myocardial muscle fibers, myocardial infarction with or without coronary artery occlusion, pulmonary congestion and edema.[3,15] Sir William Osler[25] demonstrated a contracted left ventricle with dilated chambers on the right in a victim of heat stroke; it has been speculated that such findings are probably related to right ventricular damage or an increase in pulmonary vascular resistance. Unfortunately, this is as far as this concept has gone to date. The phenomenon that clinicians have blamed for years, namely pooling in the vasodilated tissues, is probably not the cause of this cardiovascular syndrome.

Renal Aspects of Heat Stroke: Acute renal failure is seen in 25% to 35% of patients with the exertional type of heat stroke; it occurs in only 5% of patients with sedentary heat stroke.[3] There are five proposed mechanisms: direct thermal renal injury, cellular hypoxia secondary to either decreased renal blood flow or elevated metabolic demand, pigmenturia due to rhabdomyolysis or hemolysis, hyperuricemia from increased metabolism and rhabdomyolysis, and intravascular thrombosis.[3,5,12,26,27] Another interesting phenomenon has been described. In World War II in the Libyan desert, military recruits exposed to prolonged periods of heat stress demonstrated polyuria of up to 8 L/day with no real explanation.[28] It wasn't until years later that Sobel[29] demonstrated peculiar changes in renal tubular epithelium with hypokalemia and polyuria. An explanation for the polyuria is possibly hypokalemic nephropathy. At least it fits well at present with some clinical problems encountered and the deficits of potassium that occur in heat stroke, particularly the exertional form.

Gastrointestinal Aspects of Heat Stroke: Because of poor perfusion, abnormalities of electrolytes, and intravascular coagulation, frank gastrointestinal ulceration and serious hemorrhage can occur.[15] Hepatic damage is frequent; serum bilirubin may rise after several days but transaminases do not rise to their peak levels until sometime toward the end of the first week.[30] The frequency of jaundice has been argued in the literature for some time; it was described by Herman and Sullivan[31] in only 10 out of 244 patients with heat stroke. In their study of Bantu goldminers in South Africa with heat stroke, Kew and his colleagues[32] found central lubular hepatocellular degeneration and necrosis with cholestasis.

Pulmonary Aspects of Heat Stroke: Hyperventilation is common with resulting respiratory alkalosis. These patients have increased oxygen demands, so they should receive oxygen early. Careful watch must be kept for the evolution of pulmonary edema.

Hematologic Aspects of Heat Stroke: The mechanisms of the bleeding diathesis seen in some cases are basically six in number.[11,23] They are thermal inactivation of clotting factors, hepatic necrosis with impaired production of those factors, thermal destruction of platelets and megakaryocytes, damage to vascular endothelium, increased capillary permeability, and a release of substances that cause a DIC-like picture if not frank DIC at times.[3,6,15,27]

Prognosis: Mortality ranges from 17% to 70%[22,33]; 80% of deaths occur in victims over 50 years of age. The high rate of mortality from heat stroke in football players is second only to head and neck injuries.[34,35] The mortality correlates best with the product of intensity and duration of the hyperthermia, with duration being the more important.[3,27] In the Kansas City study of the 1980 heat wave, a mortality of 18% was reported. The analysts found associated disease in 65% of the victims, with hypertension, diabetes and alcoholism leading the list. Medications known to predispose to heat stroke were being used by 56% of the stricken people. Basically there are five or six drugs that produce molecular hyperactivity and hypermetabolism, that can impair thermal regulation by reducing dissipation of heat and blocking cardiovascular compensations. In the study, five prognostic factors were noted that could be used to predict probable demise. They were extreme hyperthermia (greater than 108°F or 42.2°C,) coma especially if prolonged, hypotension, need for ventilatory support, and coagulopathy.[17]

Treatment: The first step in treatment is the most important—rapid cooling.[17,23] It should be started at once at the site of exposure. Ice should be placed primarily on the neck, the axillae, and groins. Oxygen (100%) must be started as soon as possible to meet the hypermetabolic needs, with frequent examination made to detect the possible development of pulmonary edema. On arrival at an ER, rapid cooling must be urgently continued. There are several means of achieving it. Immersion in ice tubs is to be avoided, if possible, because of the inherent difficulty in handling the patient if cardiopulmonary resuscitation and cardioversion is required. Cooling the patient with sheets wet with ice water and enhancement of evaporative cooling by fanning is an effective alternate.

Weigert and Khogali[36] in the Middle East have observed a large number of heat strokes in Moslems trekking to Mecca, and managed them with a unique body cooling unit. The patient is stretched out nude in a mesh net that looks something like a hammock. Hot air at 113°F to 118°F (45°C to 48°C) is blown on the anterior and posterior surfaces of the suspended patient. The hot air is theorized to cut down on the vasoconstriction from cold which could inhibit heat dissipation. It is difficult, however, to prove the rationale of the method. Dr. Ronald Lawrence, president of the American Medical Joggers Association, says that the early vasoconstriction in response to cold only lasts about 30 seconds, so that it is not of great consequence in abrupt cooling.[37] The problems with the body cooling unit described is that it is expensive, it is not available in the United States, and it has not been proven to work any better than what we have been doing in our ER for some time.

Other cooling methods include gastric lavage with iced saline and injection of cooled IV fluids. Regardless of the method or combination of methods, the sooner the temperature falls the better. The ideal objective is to drop it to normal in an hour. Active cooling is halted when the core temperature gets to 102°F (38.8°C).[3,12,14,17] Aspirin is not to be used; it is ineffective and can worsen a bleeding diathesis. Dantrolene has been used in some cases, when the temperature would not fall with active mechanical cooling and when there was primarily increased muscular activity.[38]

In the treatment of hyperthermia the question of what to do if the patient begins to shiver during active cooling is often debated. Most of those knowledgeable in the field recommend chlorpromazine in a dose of 12.5 to 25 mg IV slowly.[15,20,27] However, there are several hazards to use of this drug. As an alpha-adrenergic receptor blocking agent, it can worsen hypotension; it can cause arrhythmias, decrease seizure threshold, and worsen the overall chances of getting rid of the heat from the body. Thus, unless the shivering actually blocks the cooling process, the drug should probably not be used.[13,23,27]

Table 2 is a list of the common complications of heat stroke.[6] With constant monitoring, good supportive care and rapid identification, those adverse effects can be avoided. A few examples may illustrate this. If cerebral edema is present, mannitol, usually 1 gm/kg IV, is indicated. Dexamethasone has been used to decrease cerebral edema in an initial dose of 4 to 10 mg IV, then in increments of 4 mg every six hours. Seizures are managed with the usual medications. The serum glucose must be checked—D50 IV may be needed. High flow oxygen and intubation may be needed if blood O_2 is low. Attention must be paid to pulmonary toilet and steroids considered if aspiration occurs. Average fluid volume replacement ranges from 1,200 ml to 1,400 ml per four-hour period but care must be used to avoid pulmonary edema.[23] If in doubt, monitor pulmonary artery pressure with a Swan-Ganz catheter. When fluid is given rapidly in hyperthermia, with quick cooling and return of vasoconstriction, pulmonary edema can be produced quite easily. If the patient demonstrates a low cardiac output, a beta-adrenergic receptor agonist may be desirable; dobutamine is the preferred drug. Drug-induced vasoconstriction should be avoided, because it can inhibit heat dissipation; atropine should not be used as it inhibits sweating.[30] If symptomatic bradycardia occurs it may be an indication for isoproterenol hydrochloride, or even right ventricular transvenous pacing. A nasogastric tube may be needed to prevent or treat gastric atony and prevent pulmonary aspiration from regurgitation. The gastric pH must be kept greater than 4 to prohibit the development of a stress ulcer. Urinary output must be maintained at 30 ml/hr; if rhabdomyolysis is present the rate of output should be higher with the addition of mannitol and sodium bicarbonate. If bleeding occurs, the underlying cause should be treated with the appropriate blood component. Heparin has been used successfully when DIC has occurred, but only in a few cases.[3,27,39]

The best treatment, as with any illness, is prevention. Those involved with team sports or jogging should educate themselves on the prevention of heat illness. Weighing before and after prolonged exercise is important. An athlete should not lose more than 5% of his body weight during exercise.[1,8] A large supply of cold water should be available. If it is palatable, it is absorbed faster.[8] The athlete with fever should avoid exercise, since he is more prone to heat stroke. Lastly, if there is a playing surface that stores heat, such as artificial turf, it should not be used in extremes of heat, or if it is used, it should be watered down frequently.[1]

We can educate the young and their direct supervisors to prevent deaths from heat illness, but the elderly are going to continue to die unless we provide a cool environment for them during heat waves at a relatively small cost.[40] Prevention is the key to reducing heat-related illness and deaths. Fortunately after the heat wave that struck Memphis in 1980, a local Thermal Emergency Operations Group was founded to address the educational needs of the public relative to

TABLE 2*

COMPLICATIONS OF HEAT STROKE

Convulsions
Bleeding diatheses
Aspiration pneumonia
Bronchopneumonia
Pulmonary embolism
Sepsis
Cardiac failure and pulmonary edema
Myocardial infarction
Cerebrovascular accident
Renal failure
Hepatic failure
Rhabdomyolysis
Neurological deficits
Gastrointestinal ulcers

*Modified from Stine.[6]

protection from excessive heat or cold. It works well.

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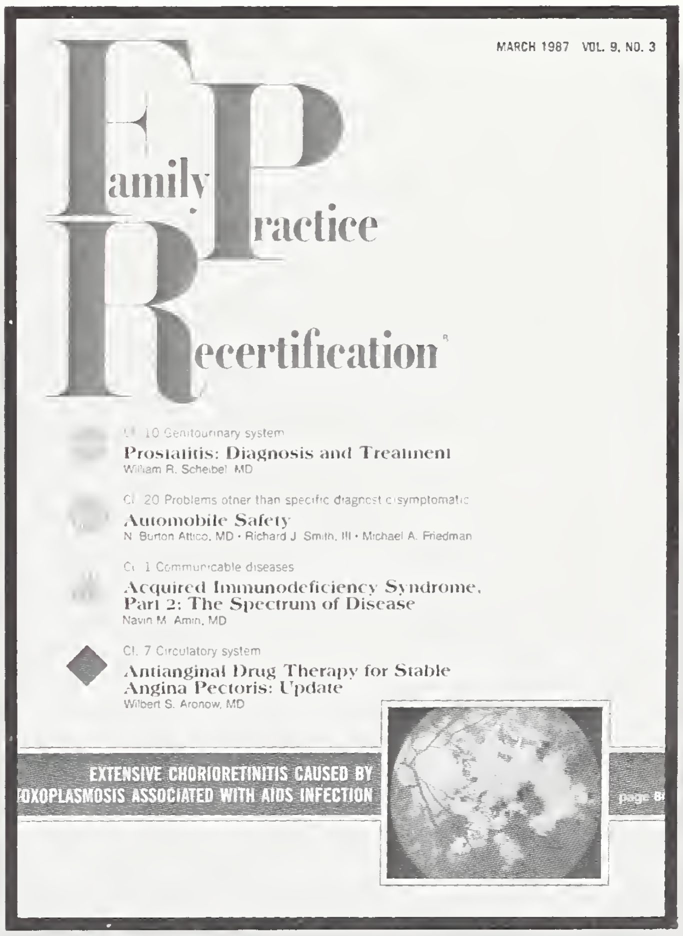

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EXTENSIVE CHORIORETINITIS CAUSED BY TOXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION

page 8

Fatal Pneumococcal Pneumonia

Case Report

A 65-year-old black woman was brought to the emergency room because of shortness of breath and confusion. The patient had been chronically ill but stable until one week earlier when she developed coryza, mild resting shortness of breath, and orthopnea. She denied cough, sputum production, hemoptysis, or fever. Six hours prior to admission her shortness of breath worsened suddenly and she was transported by ambulance to the emergency room. Her past medical history was significant for adult onset diabetes mellitus resulting in diabetic retinopathy, proteinuria with renal insufficiency (creatinine 5 to 6 mg/dl), and peripheral neuropathy. She also had hypertension with left ventricular hypertrophy and three vessel coronary artery disease. A 25-year history of smoking two packs of cigarettes per day had resulted in resting hypoxemia. She had received the 14 serotype polyvalent pneumococcal vaccine earlier.

On arrival, physical examination was remarkable for an obese elderly black woman in marked respiratory distress and sweating profusely. Her respiratory rate was 30/min, pulse 120/min, blood pressure 200/95 mm Hg, and rectal temperature 100.6° F. She was coughing up pink frothy sputum. Chest examination revealed diffuse bilateral rales to the apex of both lungs. There was no jugular venous distention, but peripheral edema was noted. Cardiac examination was obscured by loud breath sounds, but the PMI was laterally displaced.

Arterial blood for gases was obtained with the patient breathing room air. Minutes after arrival the patient suffered a respiratory arrest and was intubated and placed on a mechanical ventilator. Initial laboratory studies for blood gases revealed a pH of 7.19, Pco_2 of 53 mm Hg, and Po_2 of 29 mm Hg. The white blood cell count (WBC) was 13,900/cu mm, with a marked predominance of granulocytes. The hematocrit was 26.8% and the platelet count was normal. Sodium was 138 mEq/L, potassium 5.3 mEq/L, chloride 106 mEq/L, and bicarbonate 18 mmole/L. The blood glucose was normal, but the BUN and creatinine were elevated at 73 mg/dl and 9.0 mg/dl respectively. Electrocardiogram showed sinus tachycardia, with a new right bundle branch block and nonspecific T wave changes. Urinalysis showed 4+ proteinuria. Chest radiograph (Fig. 1) demonstrated bilateral perihilar interstitial and alveolar infiltrates, a left pleural effusion, and cardiomegaly, consistent with the clinical diagnosis of cardiogenic pulmonary edema.

The patient was admitted to the intensive care unit, where blood, urine, and sputum cultures were obtained. Gram stain of the sputum demonstrated numerous Gram-positive diplococci. Intravenous erythromycin was begun because of the suggestion of an atypical pneumonia raised by the radiograph, and the Gram staining of organisms compatible with pneumococcus. Because of continued lethargy despite correction of hypoxemia and acidosis, a lumbar puncture was performed and found to be normal. Serial cardiac enzymes failed to show evidence of an acute infarction. The right bundle branch block seen on EKG resolved with improved oxygenation, but over the next several hours the patient's status declined, with worsening hypoxemia, and hypotension with poor lung compliance. Placement of a flow-directed pulmonary artery catheter demonstrated a normal pulmonary capillary wedge pressure, low systemic resistance, and low cardiac output. The patient was treated with intravenous inotropic drugs, increasing fractions of inspired oxygen, and positive end expiratory pressure (PEEP), but remained hypotensive, hypoxemic, and acidemic. In spite of aggressive support, she died within 24 hours of admission of refractory metabolic acidosis and hypoxemia. On the day of death sputum culture and all blood cultures were reported to be growing type 14 *Streptococcus pneumoniae*.

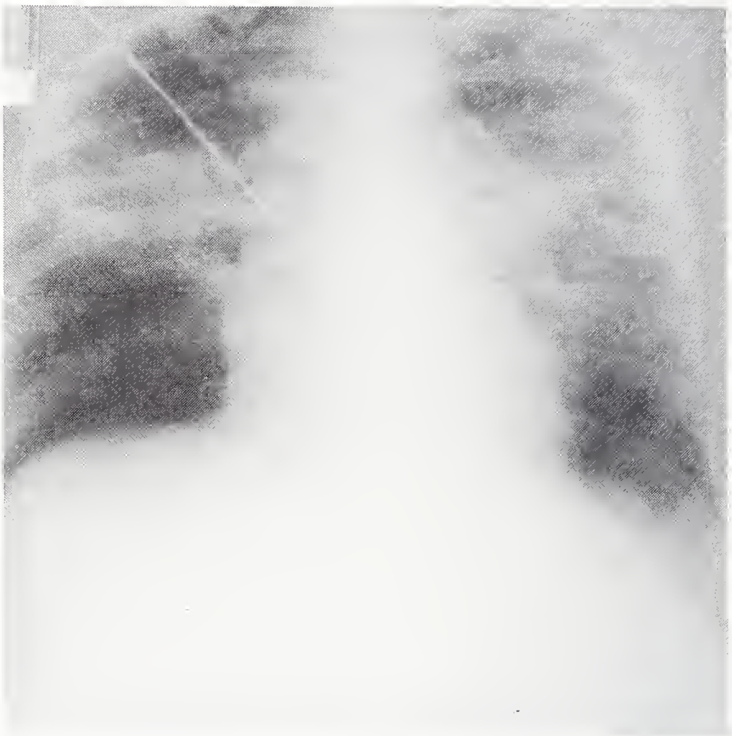

Figure 1. Portable AP chest radiograph demonstrating diffuse bilateral interstitial and alveolar infiltrates.

Discussion

This patient presents several important points for discussion. First, the initial impression of cardiogenic pulmonary edema was in error. Although physical signs and radiographic findings were compatible with the diagnosis of cardiogenic pulmonary edema in a patient who was predisposed to this condition, pulmonary wedge pressure measurements demonstrated that her pulmonary edema was from a non-cardiogenic source (ARDS). The sputum Gram stain, low grade fever,

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Prepared by Arthur P. Wheeler, M.D., Hugh J. Morgan chief medical resident, Vanderbilt Medical Center, Nashville.

white blood cell count, and hemodynamic data obtained from the pulmonary artery catheter all raised the suspicion of a pulmonary infection. Although retrospectively penicillin would have been the drug of choice for this patient, the uncertainty of the diagnosis, with unusual pulmonary infiltrates, dictated antibiotic therapy for pneumonia caused by "atypical" organisms.

Austrian and Gold[1] have demonstrated that antibiotic therapy has not decreased the mortality of patients with bacteremic pneumococcal pneumonia who die within 24 hours of admission. Similarly, it has been shown that even with the advent of sophisticated life support technology, including mechanical ventilation, PEEP, and pulmonary artery pressure monitoring, mortality from bacteremic pneumococcal pneumonia has not been decreased, but only delayed.[2] Another interesting finding is that though this patient had been immunized with the 14 serotype polyvalent vaccine for pneumococcus, she developed an infection with one of the organisms contained in the vaccine; the pneumococcal vaccine is useful but not a panacea. Most studies have demonstrated approximately 80% efficacy in the elderly, with lower rates of effectiveness in populations with severe immune deficiency.[3]

Recent reports continue to question the efficacy of pneumococcal vaccination in certain subgroups. The mortality rate of pneumococcal pneumonia for all patients remains near 20%, but rates of greater than 50% have been reported in patients with pneumonia and pneumococcal bacteremia.[1,4] The prognosis is worse with bacteremia if the patient is older than 60 years, or has multilobar pneumonia, diabetes, heart failure, or obstructive lung disease. Because many of the signs and symptoms of pneumonia seen in younger patients are minimal in the elderly, there should be a high degree of suspicion for infection in any elderly patient with a pulmonary infiltrate.

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Alcoholic Acidosis

Case Report

A 67-year-old white man with a past medical history of alcoholism, squamous cell carcinoma of the larynx, and obstructive lung disease (COPD) was admitted with a chief complaint of progressive shortness of breath for two days, not associated with cough sputum production, fever or chills. He denied orthopnea, wheezing, chest pain, or hemoptysis. He had been vomiting for two days prior to admission, with little or no food intake, but had continued to consume one pint of liquor per day. He denied the ingestion of ethylene glycol, methanol, moonshine, or other drugs.

Physical examination showed a tremulous elderly man with a pulse of 112/min, a respiratory rate of 36/min, and a blood pressure of 90/60 mm Hg. On standing, his systolic blood pressure fell by 10 mm Hg, with a rise in heart rate. There was no evidence of jugular venous distention, or abnormality of his tracheostomy stoma. The lung fields were clear to percussion and auscultation, and the cardiac examination revealed no murmur or gallop rhythm. The liver and spleen were of normal size, and no stigmata of alcoholic liver disease were noted. The extremities did not show clubbing or edema.

On laboratory examination, arterial blood gas studies showed a pH of 7.4, a Pco_2 of 18 mm Hg, and a Po_2 of 93 mm Hg on room air. Sodium was 137 mEq/L, potassium 4.2 mEq/L, chloride 99 mEq/L, and serum bicarbonate 15 mmole/L, with a calculated anion gap of 23. Serum albumin was 3.7 gm/dl. Serum glucose, creatinine, and BUN were all normal, as was a calculated osmolar gap. Serum ketones were present in a titer of 1:8, and urinalysis showed 4+ ketonuria. Serum lactate was 4.0 mEq/L (normal 0.5 to 2.2). Chest x-ray was remarkable only for changes of COPD.

Discussion

This patient illustrates the complexities of acid-base and electrolyte disturbances associated with excessive alcohol consumption. Patients with "alcoholic acidosis" frequently have a metabolic acidosis composed of ketoacidosis and lactic acidosis as a result of the effects of starvation and alcohol on intermediary metabolism.[1-3] Poor tissue perfusion induced by volume depletion also contributes to production of lactic acid. Vomiting occurs in a sizable percentage of these patients, leading to a mixed metabolic acidosis and metabolic alkalosis. When respiratory alkalosis due to alcohol withdrawal or pneumonia is superimposed, a triple acid-base disorder may result.

The cellular biochemistry of alcoholic acidosis is interesting and complex. Insulin levels are decreased in alcoholic ketosis, leading to increased lipolysis, increased serum-free fatty acids, and the formation of ketone bodies. Altered oxidative processes in the liver combined with low insulin levels also lead to a decrease in hepatic gluconeogenesis. Nausea and vomiting are frequent, often resulting in hepatic glycogen depletion and hypoglycemia.[4] Decreased insulin levels also promote lactate formation from glucose metabolism, rather than its usual complete oxidation via the Krebs cycle. Growth hormone and cortisol are increased, and also increase ketone production through lipolysis. The usual metabolism of ethanol through acetyl coA to the Krebs cycle is diverted to the production of ketoacids.

The mainstay of treatment in alcoholic acidosis is intravascular volume replacement.[5] Total body fluid deficits are similar to those seen in diabetic ketoacidosis (up to 6 liters). The administration of sodium bicarbonate is not necessary except in the presence of severe acidosis (pH < 7.1). The administration of intravenous glucose in solution at a rate of 7 gm/hr has been shown to accelerate the resolution of the acidosis. Patients with alcohol-related acidosis and vomiting frequently have potassium depletion due to the secondary effects of volume depletion and subsequent hyperaldosteronism. Insulin does not accelerate resolution of the acidosis and is therefore not recommended.[5] The typical course is resolution to the acidosis within 12 to 24 hours.

The level of serum ketones as determined by the nitroprusside test may be misleading. Because of alterations in acid-base status, a particular ketone body, beta-hydroxybutyrate, is the predominant ketoacid seen in alcoholic acidosis. Beta-hydroxybutyrate is not detected by the nitroprusside reaction, but is in equilibrium with acetoacetate, the predominant ketoacid that is detected by this method. Severe acidosis favors the production of beta-hydroxybutyrate over other ketoacids. Therefore, as the metabolic acidosis improves, serum ketones may paradoxically appear to increase as beta-hydroxybutyrate is converted to acetoacetate. As in any patient with chronic alcohol abuse, the administration of thiamine, B vitamins, and balanced nutrition is important adjunctive therapy. Close monitoring for the development of alcohol withdrawal syndrome is also prudent.

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Trauma Rounds

Myocardial Contusion

GREGORY G. STANFORD, M.D.; EUGENE C. MANGIANTE, M.D.; and
TIMOTHY C. FABIAN, M.D.

The diagnosis and management of myocardial contusion continues to plague the physician dealing with trauma. There is no diagnostic test for myocardial contusion, and there is no standard to evaluate new methods of detection and diagnosis. It is important to determine the presence or absence of cardiac injury, since if left untreated it can lead to sudden death, heart failure, fatal arrhythmias, coronary occlusion, and cardiac tamponade. Because patients with myocardial contusion frequently have multiple injuries requiring surgical treatment, its presence is all the more significant, since such patients may require more sophisticated and invasive monitoring during general anesthesia.

Case Report

A 26-year-old white woman was involved in a single vehicle accident in rural Arkansas about six hours prior to admission to the Presley Trauma Center. Following a difficult extrication from the vehicle she was transported to a local hospital, where she was found to have normal vital signs but was confused and somewhat combative. She was then transported to the Presley Trauma Center. On arrival she was lethargic and intermittently combative, with a heart rate of 117/min with blood pressure of 134/87 mm Hg. She had a deep chin laceration and a large ecchymosis over her left anterior chest. Breath sounds were clear, and her heart sounds were normal without a murmur or rub. Her abdomen was remarkable only for tenderness over the pubis. There were no extremity fractures. The hematocrit was 30%, with a white blood count of 27,700/cu mm. Urinalysis revealed 100 to 150 RBC/HPF. The electrocardiogram showed T wave inversion in leads V_1 through V_3 but was otherwise normal. The chest film showed bibasilar pulmonary contusions, pneumomediastinum, left chest wall subcutaneous emphysema, and a right hemothorax. The aortic knob was sharp. Pelvic films revealed fractures of both inferior and superior pubi rami bilaterally, with bilateral comminuted acetabular fractures. The CT examination of the head was normal; a CT of the abdomen and pelvis showed some asymmetry to the left kidney, multiple pelvic fractures, and a large associated pelvic hematoma. Distortion of the bladder by the hematoma without evidence of damage to the bladder itself was seen on cystogram. Peritoneal lavage fluid contained 100 WBC/cu mm and 31,000 RBC/cu mm.

The patient was placed in the Trauma Intensive Care Unit for observation. Bilateral thoracostomy tubes were required for her right hemothorax and left pneumothorax. Cardiac isoenzyme studies showed a CPK-MB of 422 IU/L (12%), which fell after six hours to 203 IU/L (4.5%). She experienced no arrhythmias or myocardial failure. Serial electrocardiograms showed no evolution, with persistent T-inversion in leads V_1 through V_3. A decreased right ventricular ejection fraction (30% to 40%) with hypokinetic wall motion and a normal left ventricle was seen on gated cardiac study. A repeat study seven days later was normal. She had a long hospital course complicated by persistent disorientation, which slowly resolved over six weeks. Her pelvic fractures were treated with bilateral tibial traction pins and skeletal traction for eight weeks. She was discharged home ten weeks after admission.

Discussion

The typical patient sustaining a myocardial contusion has been involved in a motor vehicle accident in which the upper torso has struck the steering wheel or the dashboard. The accident itself may not be of great magnitude, several cases of myocardial contusion having been documented after deceleration from velocities of less than 20 miles per hour. The pathophysiology relates to the heart being compressed between the sternum and the vertebral column. Another suggested mechanism is rapid abdominal and lower extremity compression, which upwardly displaces abdominal viscera leading to cardiac injury. Any moderate force applied to the anterior chest wall, including chest wall crush, tangential gunshot wounds, athletic injury, agricultural accidents, and external cardiac massage, can produce myocardial injury. The incidence of myocardial contusion varies from 9% to 76% when liberal diagnostic criteria are applied, but most recent series report an incidence of from 15% to 30% in patients sustaining blunt chest trauma.[1-3]

The most common symptom of myocardial

From the Presley Trauma Center and the University of Tennessee College of Medicine, Memphis.

contusion is precordial pain not unlike that associated with an acute myocardial infarction; tachycardia is the most frequent physical sign. Other findings include dyspnea, tachypnea, gallop rhythms, and jugular venous distension, which may indicate low cardiac output. Unfortunately, all the symptoms and physical signs are nonspecific and difficult to interpret in the face of multiple other injuries. Patients with flail chest, liver injury, splenic injury, or pelvic fractures have a significant increase in cardiac complications.

The electrocardiogram may show many significant changes, none of which is specific for myocardial contusion. The ECG tends to reflect abnormalities of the left ventricle, and is relatively insensitive to injuries of the right ventricle which is more commonly damaged in blunt cardiac trauma due to its anterior location just below the sternum. ECG changes tend to revert to normal after a variable length of time. Sinus tachycardia is the most common dysrhythmia,[3] and other supraventricular dysrhythmias such as flutter are common. Frequent premature ventricular contractions are often present, but ventricular tachycardia and fibrillation are rarely seen. In experimental preparations, ventricular dysrhythmias predominate, and are seen immediately after the injury occurs.[4] This may be the cause of immediate death from myocardial contusion, and the patients who reach treatment are those without early ventricular irritability. The ST-T segment is frequently referred to in discussion of myocardial contusion, with most authors citing nonspecific changes.[5] The importance of these changes is that resolution may be the only indication of a previous myocardial injury.

Total creatine kinase (CPK) is frequently elevated in patients sustaining blunt trauma due to associated musculoskeletal trauma. CPK consists of two subunits (M and B), which combine to form the three isoenzymes of CPK: MM, found predominately in muscle; BB, which predominates in the brain; and MB, which is found chiefly in the myocardium. Plasma MB is elevated in patients following a myocardial infarction, and accounts for 10% to 15% total CPK activity.[6] CPK-MB disappears from the circulation more rapidly than CPK-MM, and is more labile. Therefore, the peak plasma CPK-MB may be somewhat lower than the corresponding percentage of myocardial MB. Except in certain neuromuscular diseases, CPK-MB is specific for myocardial injury and is unreliable in crush injuries when the total CPK is greater than 2,000 IU.

Elevation of the CPK-MB indicates myocardial damage with leakage of CPK from damaged myocardial cells. Blunt trauma to the heart may not be significant enough to cause myocardial cell injury, but may be severe enough to cause dysrhythmias. Some authorities distinguish this difference by calling injury without cell damage a "concussion," limiting the use of the word "contusion" to myocardial damage documented by an elevation of the CPK-MB. In a recent study from the Presley Trauma Center,[7] CPK-MB by fractional electrophoresis was very reliable in predicting blunt myocardial injury. CPK-MB is therefore a reliable tool in the diagnosis of myocardial contusion in patients who do not have specific ECG changes. It is of benefit to separate those patients with acute ECG changes from those with chronic ECG abnormalities.

Various noninvasive studies have been applied to the evaluation of cardiac contusions. The gated blood pool scan has been used since 1971 as a noninvasive study of cardiac function. It has since been evaluated in the diagnosis of myocardial contusion by several investigators.[2,8,9] Its usefulness is still in question, but further refinements in the technique may make it a valuable tool in the future. The technetium pyrophosphate scan, which is useful in evaluating myocardial infarction, appears too insensitive for evaluation of blunt cardiac injuries[5]; until further studies are done it cannot be recommended routinely.

The echocardiogram may be useful under certain circumstances. Frazee et al[10] have declared that the 2-D echocardiogram is required to make the diagnosis of a myocardial contusion, with patients having a normal study but other evidence of myocardial injury being classified as having a myocardial concussion. The 2D echocardiogram was found to be abnormal as often in patients without evidence of myocardial injury as in those with myocardial injury established by ECG or CPK criteria.[8] It appears that refinements in the technique may offer hope in the future, but it cannot be recommended routinely at this time in the evaluation of possible myocardial contusion.

Myocardial performance has been evaluated by several authors following myocardial contusion. Torres-Mirabal[2] constructed myocardial performance curves in nine patients with myocardial contusion. All nine patients have biventricular or univentricular dysfunction. Six patients had biventricular dysfunction, two had isolated right ventricular dysfunction, and one had isolated left ventricular dysfunction. In three of the nine patients ventricular function returned to normal within four days. In another study,[1] 11 of 19 patients with a myocardial contusion required inotropic support because of inadequate cardiac output. One patient required intra-aortic balloon counterpulsation to maintain an adequate cardiac output.

Many patients sustaining blunt trauma require general anesthesia for repair of associated injuries. It has not been determined if patients with a coexisting myocardial contusion are at a greater risk for morbidity and mortality than those without myocardial contusion. In a study from the Shock Trauma Center in Baltimore,[1] 19 patients with myocardial contusion underwent emergency surgery for associated injuries; 11 required perioperative cardiac inotropic support, and one required intra-aortic balloon support. No morbidity or mortality was attributed to the cardiac contusion in these patients. In the study done at the Presley Trauma Center,[7] operative procedures were performed on 37 patients with documented myocardial contusion, with no increase in morbidity or mortality. Emergency surgery is not contraindicated in patients with a suspected myocardial contusion, but careful attention must be paid to the cardiac status, including invasive monitoring when necessary to detect and treat dysrhythmias and cardiac insufficiency.

At the Presley Trauma Center, all patients with severe chest trauma are treated as though they have myocardial contusions. Electrocardiograms are obtained upon admission and daily for three days if normal, or until abnormalities clear. CPK-MB isoenzymes are determined at admission and every eight hours for 36 hours; all CPK determinations are done by electrophoresis. These patients are monitored for three days or until the cardiac rhythm is stable. Pulmonary artery catheters are used liberally in patients with any evidence of cardiac decompensation. Emergency procedures are performed when necessary, using inotropic agents and antiarrhythmic agents to maintain hemodynamic stability. We have not found intra-aortic assist devices to be necessary, but they can be used when cardiac decompensation does not respond to inotropic support. Most of these patients do well, without sequelae of their cardiac injury, if attention is paid to early cardiac monitoring and complications are treated appropriately.

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Loss Prevention Case of the Month

Close Counts Only in Horseshoes

J. KELLEY AVERY, M.D.

Case Report

The patient, a 30-year-old nurse, came to her primary care physician complaining of numbness in her right knee and toes, which had developed fairly suddenly about three months before. She remembered the day the symptoms developed; there was no history of pain or trauma. The primary care physician referred her to a neurosurgeon.

A detailed neurologic examination was recorded in the outpatient medical record including the history of a "lazy eye" on the left, with confirming physical findings, some motor spasticity in the lower extremities, worse on the right, temporal pallor of the optic disk, symmetrical hyperactive deep tendon reflexes upper and lower extremities, absent Babinski's signs bilaterally, and lack of superficial abdominal reflexes. The neurosurgeon's impression was "possible demyelinating disease." Mild sedation was prescribed and the patient was to return for reevaluation in ten days.

At this second visit her legs were "improving" and the nurse thought that she could return to work in "ten days or so." She did return to work approximately three weeks after her initial evaluation, but three days later again consulted the neurosurgeon complaining of "dizziness." On this occasion, she was admitted to the hospital where further workup, including evoked potentials, cerebrospinal fluid examination, and IgG and myelin-based protein studies were done. The results of these tests, along with continued physician evaluation and observation, were suggestive of a demyelinating disease, and the patient was discharged with the diagnosis of "probable MS."

Two months later the neurosurgeon noted that his patient was now "ambulatory with cane." Six months after the initial visit to the neurosurgeon and nine months after the development of symptoms, the patient reported to another physician, requesting a second opinion regarding the diagnosis of multiple sclerosis. Again, extremely thorough evaluation, medical history, physical examination, and results of tests done by the first neurosurgeon were found to support the diagnosis of multiple sclerosis, but he believed that even in the absence of any history of trauma, CT of the spine should be done. This examination revealed a lesion at T9 and T10, and thoracic and lumbar myelograms revealed a "partial block at T9 and T10, predominately extradural." Following the myelogram, the patient showed some neurological deterioration, and an emergency thoracic laminectomy was done, revealing a herniated intervertebral disk at T9 and T10. The patient is now paraplegic.

Loss Prevention Notes

Every physician has had the experience of very carefully evaluating a patient by physical examination and history, arriving at an impression and proceeding to order appropriate studies designed to confirm his initial impression. This might be likened to focusing so intently on a single tree that one loses sight of the forest. This appears to characterize the trap this physician fell into in dealing with this particular patient. The initial very careful history and physical examination certainly pointed toward a demyelinating disease, and considering the sex and age group, the preliminary diagnosis of multiple sclerosis was extremely logical. As the laboratory and other clinical studies progressed, all seemed to confirm the initial impression.

A very important consideration in our management of patients is cost control. We are increasingly aware of the financial burden we place on our patients when advanced technologic procedures are employed that cost a lot of money. This may have influenced our physician in his failure to order other tests on his patient earlier in her course.

There are few areas in medicine where a wrong conclusion, no matter how logical, drawn from the information at hand, can be as devastating as it is in the specialties of neurology and neurosurgery. Even though this patient seemed to be following a very predictable course with the initial diagnosis of multiple sclerosis, in retrospect it would have been extremely valuable for our physician to have consulted a colleague in his own field. As medical litigation becomes increasingly important in our lives, and as awards in medical malpractice continue to escalate, we need to be-

(Continued on page 298)

Dr. Avery is medical director for Continuing Medical Education, St. Thomas Hospital, Nashville, and chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood.

Reducing WIC Formula Costs To Allow Services for More Eligible Tennesseans

LEE FLESHOOD, Ph.D., M.P.A.; JANE BAXTER, M.S., R.D.;
MARK HARLAND, B.S.; and MANNY MARTINS, M.P.A.

What Is WIC?

The Special Supplemental Food Program for Women, Infants and Children (WIC) serves about 75,000 low income pregnant, breastfeeding and postpartum women, infants, and children at nutritional risk in Tennessee. Eligible persons certified for WIC receive nutrition education, specific nutritious foods to supplement their diet, and health care service or referral. The overall purposes of the program are to prevent the occurrence of health problems and improve the health status of participating mothers and children.

The supplemental foods provided by WIC are specific, and were selected because they are high in the nutrients that contribute to an adequate diet for the participants' nutritional needs: protein, iron, calcium, vitamins A and C. These are the nutrients most likely to be lacking in the diets of this population group. Infants who are bottlefed may get iron fortified formula, as well as infant cereal and juice after 6 months of age. Women and children get milk, cheese, cereals high in iron, fruit juice, and dried beans when in dietary need. These foods are intended to be a supplement to the daily diet, not to provide total food needs.

Through nutrition education, participants are helped to know what is in a good daily diet and how to use the WIC food package as a part of their daily food needs. The supplemental foods are prescribed by the certifying health professional according to the individual's age, nutritional need, and food preparation and storage facilities.

Who Does WIC Serve?

Of the 75,000 people WIC serves, 19,500 (26%) are women, 23,500 (32%) are infants, and 32,000 (42%) are children.

Eligibility is based on the following criteria:

- resident of area served by health department clinic in one of the 94 counties served by WIC (the state operates only the Commodity Supplemental Food Program [CSFP] in Dyer County, and CSFP and WIC in two other counties).
- income at or below 170% of the Secretary of Agriculture's poverty index. For a family of two, the income eligibility guideline is currently $12,308 annually.
- have a diagnosed medical or nutritional problem such as low birth weight, anemia, growth stunting, overweight, or high risk pregnancy.
- be a pregnant, lactating, or high risk postpartum woman, or an infant or child up to age 5 who meets the above criteria.

A certification period lasts to the age of one year for infants, six months for children, and six weeks past the birth of the infant for a woman. Breastfeeding women can be certified for up to one year as long as they continue to breastfeed.

Outcome evaluations are showing that WIC participation is associated with fewer low birth weight infants, reduced anemia, and stunted growth or obesity in participants. WIC is 100% federally funded and is authorized under Public Law 95-627. The WIC Program is open to all el-

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From the Nutrition Services Division, Health Services Administration, Tennessee Department of Health and Environment, Nashville.

igible persons regardless of race, color, national origin, age, sex, handicap, or religious or political belief.

How Does the Participant Receive the WIC Foods?

After being determined eligible, vouchers are issued for a specific food package. The vouchers can be redeemed at one of the more than 1,300 merchants certified to receive them. A few states have a direct warehouse or a home delivery system, which substantially reduces front end costs for formula. Most states operate a voucher system similar to Tennessee's. Maintaining the retail voucher system prevents some of the service delivery and patient access problems inherent in a direct distribution system.

Impact of Increasing Formula Costs

Between 1978 and 1986, infant formula wholesale prices from the three major manufacturers more than doubled. This increase was much greater than for the other WIC foods. Consequently, the proportion of WIC food dollars Tennessee spends on infant formula has continued to grow (over 37% in 1986).

The Tennessee WIC Program purchases approximately one-third of all the infant formula sold in the state. In 1986 wholesale formula prices rose approximately 11%, thereby increasing the formula costs to Tennessee by over $700,000 in one year.

Tennessee's Solution to Increasing Formula Costs

Beginning in early 1984, the process was initiated that resulted in a contract with Wyeth Laboratories. Through the state competitive bidding process, a price enhancement (rebate) of $0.48 per can of infant formula was received, and starting June 1, 1987 SMA or Nursoy will be the standard WIC formula for the contract period. Under this system, mothers with WIC vouchers will be permitted to buy only the milk or soy based formula designated on the WIC vouchers. Exceptions will be made on the basis of medical need; infants who do not tolerate the designated brand will be able to switch formulas with a prescription from their physician. The prescription should state the type of formula required and the existing medical reason/condition necessitating the formula requirement for the particular infant, and must be signed by the physician.

The price enhancement to the program from the formula company will be used to increase participation. The Tennessee WIC Program is currently able to serve approximately 52% of all the women, infants, and children eligible for WIC services. It is expected that the annual savings will be about $4 million and will allow services to an additional 8,000 eligible people.

This innovative solution to the high cost of formula could serve as a national model for other states using a voucher delivery system.

Loss Prevention Case of the Month . . .

(Continued from page 296)

come increasingly sensitive to our patients' dissatisfaction with the course of their illness. In this case, one wonders if the patient gave any indication to our attending physician that she was not satisfied with his management of her case. Had she, he could have called for a consultation, and in all probability, even if the results were unchanged, the litigation could have been avoided.

Perhaps we could also conclude from this case that when we are dealing with a situation where consequences of a wrong conclusion are so devastating, our patients would be better served and some bad results prevented if we cast aside all thoughts of cost containment and used all the technologic tools available to rule out other possible diagnoses.

JAMES T. GALYON

Should We Sanction PRO?

With government money comes government controls and regulations. Shortly after the federal Medicare money became available, review programs such as PSRO and the PRO were developed to contain costs, flying under the false flag of quality control. Recently, with the implementation of DRG programs, there have been so many complaints to Congress and HCFA about premature discharges and other quality assurance problems that action was taken. The Consolidated Omnibus Budget Reconciliation Act (COBRA and OBRA) direct the PROs about the country to judge quality of care and to deny payment if professional standards of care have not been met by physicians, hospitals, and other providers.

All reasonable doctors would agree there are instances of medical care that do not meet the best quality standards. These may be in fact the direct result of some of the federal programs that now are attempting to sanction providers for poor quality of care. We can hardly take a position against quality assurance.

The current PRO, Mid-South Foundation for Medical Care, has incited an outpouring of protests and concern with its recent transmittals and sanctions. Several serious mistakes have been made. The PRO has failed to communicate its methods and its intentions to physicians. The avowed intention and purpose is education for quality care. Transmittals have been issued that were poorly conceived and served to exacerbate physicians' fears, distrust, and hostility. The directors have been so poorly informed by their staff and bureaucracy that they have been unable to oversee these activities and to respond to physician concerns. Lastly, the time frames directed by HCFA are unrealistic and unworkable.

On the brighter side, the chairman of the PRO Board, Kenneth Phelps, M.D., has shown considerable evidence of trying to correct these problems, within the laws and regulations imposed by HCFA. He has met with the TMA Board of Trustees and with the TMA Hospital Medical Staff Section to answer questions, to inform us of their activities, and to respond to our concerns.

There are many needs that must be met within the PRO program if it is to have a chance to succeed. First, there must be better communication from the Board to the doctors of this state. In response to this need, the PRO will be developing newsletters to the physician community. Second, the physician Board of the PRO must be in a position to review the transmittals and proposed standards *before* their circulation, to prevent such ludicrous documents as transmittal #32, which severely damaged the credibility of the PRO and its Board. Third, the PRO Board should insist through its channels to Washington that the time frames and other factors in the Quality Assurance Program be realistic and fair to physicians and ultimately their patients. Finally, the Board should seek to work within the system so long as its professional activities constructively improve quality of care.

Certain conclusions can be drawn. This is a new law and a new set of regulations, a new board, and a new professional staff. Hasty implementation has led to mistakes. A change cannot be made without considering the alternatives. Would we change to a new PRO remote from us (perhaps in Indiana) that has even less ability and inclination to respond to our needs? Would we change to a PRO composed of non-M.D.s, individuals perhaps not physician friendly?

Our best course of action at this time should be to encourage all of our colleagues to practice high quality medicine that can live up to realistic standards and still be cost effective.

The TMA will carefully monitor the actions of the PRO to develop any constructive alternatives for better quality of care and less oppressive bureaucratic interference.

We should allow time for our new PRO Board and officers to function within the law; but, if this cannot be done, then they should withdraw their names and support, rather than lend dignity and legitimacy to an impossible program. We should all work to see that this is not necessary, since the alternatives would most likely be very unpleasant.

James T. Galyon, M.D.

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MAY, 1987

editorials

In and Out and In Between

Whether one views the medical scene today dispassionately or passionately, it seems a mess. It is hard for anyone involved in it to view it dispassionately, but the only real difference would seem to be the amount of ichor it evokes, which sometimes is considerable; it often flows out over pages of typescript, and sometimes engulfs editors (namely me) in the backwash. Whether one is being dispassionate or not seems to be largely a matter of perception (bias is what the other fellow has). The only way to avoid it is to keep quiet.

Viewing the mess, I get the impression that cleaning it up rivals trying to unscramble eggs. The main problem seems to be that everyone is looking for (or at least hoping for) a simple answer, where there is none. In the first place, the problem itself is not a simple one; it is not even a *complex* one. It is rather a whole host of complex ones. Some see it (or them) as nothing lots of money would not cure. Though that certainly would not hurt, it too is an oversimplification. I was castigated by one of our readers for implying (actually, I did not imply it; I *said* it) that doctors are becoming disenchanted, and leaving the ranks. They are, the writer said, still making a good living. So would they walk out on it?

All I know is, conversations such as the one reported to me the other day are being repeated with increasing frequency. My informant said that during a telephone conversation, an old school chum in another state told him, “I'm going to hang it up.” My informant observed that that was a surprise, as he didn't know he was thinking of retiring, whereupon his friend said, “Who said anything about retiring? I meant *quit*!” There is a fine distinction there, which I am sure is not lost on you. He is far from alone; more might join in if they had any other options. The pages of this issue of the *Journal* alone are rife with dissatisfaction.

Take our Mail Box. In it is an exchange between a physician in Shelbyville and the Department of Health and Environment's chief medical officer, Dr. Fredia Wadley. One of the things Dr. Wadley gets paid for is to absorb abuse, but in this case, she is stuck with defending a leaky fort. Or look at the Loss Prevention Case of the Month, written by the chairman of SVMIC's Loss Prevention Committee. His last statement simply reiterates what all practicing physicians already know—that cost-effective medicine can get you into trouble. In fact, good, cost-effective care can do you in probably as often as negligence.

The junior Senator from Tennessee was quoted in last Sunday's newspaper as laying the blame for the high cost of professional liability insurance on negligent doctors and the insurance companies. Such rhetoric sounds good in political campaigns, but it has little to do with reality. Not being privy to the finances of insurance companies, I cannot speak for them. I do know, though, that our own SVMIC ties rates to its experience, and gives rebates as its experience improves. What the Senator appears not to have considered is that doctors get sued not only for poor results due to negligence. Doctors get sued also, and patients given awards—*huge* awards—for poor results due to no one's fault at all, but simply because no good result was possible. It is indemnification for bad luck, and the deep pocket is tapped for it. To be fair, the doctor often gets sued because he failed to communicate that to the patient in the first place. That problem is due to continue so long as we retain the contingency system; that shows no signs of disappearing, as lawyers are the ones who make the laws.

It is a truism, espoused throughout our history, that the marketplace will take care of any situation, whatever it is. Doubtless it would; but politicians, for various reasons, few of them salutary, are unwilling to entrust their constituents (read *votes*) to the marketplace. They had rather manipulate it to satisfy their own special interests. And so those with services or commodities to sell become the pawns of those interests.

What we need in this country is a control on the controllers. As it stands, they can pretty much pursue their own course. Those in control, though, are not our elected representatives, but the bureaucrats in the various government agencies. Given a rough outline by the statutes, they proceed to fill in the blanks with their regulations. Frequently the legislators cannot recognize their own handiwork in the finished product. While the electorate can vote the politicians and upper echelons of the executive branch in or out, there is no surcease from petty bureaucrats, who like Tennyson's brook, run on and on forever. Because they have been around a long time, and are well informed in their own narrow field, they are the tutors of our legislators. Perhaps we should have a periodic Sabbath year, in which we throw them all out and start over.

It is sometimes said, often only out of pique, that those who can't do, teach. It seems those who cannot do either one, write and enforce regulations. They are called civil servants; they are in fact not our servants, but our masters. We are blessed with such bureaucrats in the medical profession, too. They do things like run HMOs and PROs. On the one hand, it appears we are stuck with bureaucrats in those jobs because no one else will do them; on the other hand, perhaps it is that those individuals just appear the way they do because they are in those jobs. Maybe bureaucrats are made and not born, and that like clothes, the system makes the man. In any case, under the present system, there is no escaping them.

In our dreams we remember a time when one did not have to choose up sides, and all of us practiced medicine the way we chose in all good fellowship. We wake to find that once Nashville had an Academy and Davidson County a Medical Society; only after a few well attended funerals could they merge. Wherever there has been a medical school, there has been "town and gown." Once there were several medical schools in Nashville, with the ins and the outs and those in between. Competition more often than not had, as it still has, a fiscal basis.

Each member of the human race has his own vision for his world. Each tries to mold everyone else's world according to that vision. It inevitably leads to conflict. The doctor's world is being increasingly bent out of shape by forces from above—by the bureaucrats. Only in focusing on a common goal can we hope to resist those forces. Medicine has never been very good at that, and so more often than not we are hoist by our own petard.

Alas.

J.B.T.

I Say It's Brussels Sprouts

I am forever in the debt of my grade school music appreciation teacher for exposing me to the joys of listening to the inventions of the great classical composers. I thought she was pretty classical herself—at least as old as Beethoven. Actually, she was probably no more than 15 or so years older than I, but when you are maybe 12 years old, you are centuries removed. She was, we thought, an old maid, attractive in a severe sort of way—and severe too. She was a good teacher, and despite our misgivings, I think she finally did get married in her old age—say about 35. But I digress.

My mother used to worry about my teacher's devotion to music. She was so devoted to it, my mother said, that she couldn't enjoy it. Unlike my mother, who was willing to live and let live, and be happy just to be able to go to a concert (my father had a self-confessed "tin ear"), she was forever picking the orchestra and/or artists to pieces.

I was listening the other day to an interview with that marvelous operatic soprano Martina Arroyo, who was agreeing with a quotation from another magnificent soprano, Dame Joan Sutherland, in commiserating with individuals who have perfect, or absolute, pitch. Dame Joan claims to have only relative pitch, whereas Miss Arroyo claims neither. They agreed that individuals with perfect pitch must remain in perpetual torment; Toscaninni is said to have been continually driven to distraction in his attempt to get all the woodwinds to play in tune. In truth, nothing—or at least almost nothing, in nature or out of it—is that precise. The music of the spheres is perpetually flat, as the universe is winding down—very slowly, to be sure—but still winding down, except when there is a burst of energy, such as with the creation of a super-nova, that sends a segment of it sharp. One would think that that music would have absolute pitch, but in fact it is all a little off-key. So are most of us.

Though it is too late to prove it now, I figure that my music teacher must have been distraught due to having perfect pitch. It must be hard on a critic to have perfect pitch, but it can be even harder on the critic's audience. Off and on, I listen to a program on public radio called "First Hearing," on which new renditions of compositions are aired, most often old favorites, and usually by noted artists or ensembles; the rendition is judged by a panel. Perhaps I am too easy to please. I think I have, like Dame Joan, relative pitch, since I immediately spot anything that is off-key, but I learned from my mother that unless it is habitual, not to take it to heart. I seldom hear anything on the program played or sung off-key, and I seldom hear such a criticism leveled at the works. The criticisms I hear are of nuances that are beyond my powers to recognize, and I suspect usually theirs as well, though they are quick to level charges of insensitivity, heavy handedness, the wrong tempo—you know: totally subjective things in which the tastes of the artist or the conductor are in conflict with theirs. It is interesting to me that the members of the panel frequently disagree—which in matters of taste is not unexpected. This disagreement keeps them all honest and the proceedings lively and relatively light-hearted—except perhaps as it affects the gored ox.

All of that is all very well if it is only a game, which is indeed the way I look at it. If I hear the piece played, regardless of what those critics may say I can join Dennis the Menace and say, "I say it's spinach, and I say the hell with it," or vice versa. This is not to pick on spinach, which despite Dennis' testimony, I like. I am not certain when I started liking it, because I think it is probably natural to dislike spinach. There are reasons to eat spinach, and so it is well to develop a taste for it. I suppose I could develop a taste for bourbon, which I dislike, but I see no reason to, since Scotch serves so well, and in fact I prefer Irish anyway. I will drink water in preference to bourbon. (Actually, under the proper circumstances—indeed, most circumstances—I drink water in preference to anything else. I hope you do too. But again, I digress.)

There is a difference in learning to like something and getting used to it. Toleration is still another alternative. These are all relatively positive reactions. I shall not dwell on the negative ones; not only that, I shall dwell on the others only to say that I can sleep right through most contemporary music. Some of it is too loud for sleep, and I like an occasional piece. In short, mostly I tolerate it. I don't know what the psychologists would say about it, but I believe there are sequences and combinations of sound that are naturally pleasing and others that are naturally not. I am willing to try to like contemporary pieces. I did after all learn to like spinach. On the other hand, though I will eat Brussels sprouts in a pinch, after 50 or more years of trying to change my preferences, I still detest them. Lately I have quit trying. I have not yet done that to contemporary music, though I am close. It takes more time and effort than at my age I am willing to expend.

I know people, my wife among them, who seem genuinely fond of Brussels sprouts. They say they are, and since I have friends who are incredulous when I tell them—truthfully—that turnip greens, properly prepared, are among my favorites of all of the things I put in my mouth, I have no reason to doubt them. There is after all no accounting for tastes; I therefore assume that there are indeed people who prefer the noise (in my view) that comprises much contemporary music, even though I find it hard to believe.

With all the recordings around, we need someone (critics) to tell us which ones are technically satisfactory, though if I am going to make a purchase, what with their now being so dear, I prefer to listen first. On the other hand, I do not need someone to tell me I should like a piece, when I know I don't. It is his taste against mine, and I abhor arbiters of taste.

In their appropriate place, I like tea (iced, with lemon, and hot, with milk), coffee black, buttermilk (sweet milk, too), beer, wine, and Irish whiskey—not mixed, and not necessarily in that order. I like Dixieland, Bluegrass, country western (sometimes) and classical music, also sometimes (or what *I* call classical music), including opera, again not necessarily in that order. There are Brussels sprouts in all of those, as well as in other, categories. I do not need a critic to point them out; I know them when I see them.

We have our problems in medicine, to be sure. Though I did not consider it at the time I chose medicine (I was too young, to begin with), I am grateful to be dealing more or less with tangibles, where things are generally objective (note the qualifications), and where changes are made mostly for advancement of knowledge and betterment of the human condition, and not just to be in the avant garde, where critics try to make bad things good, and good things bad when it is "chic" to do it.

J.B.T.

Drinkin' Rum an' Coca-Cola

Scattered like bright emeralds in a tie-dyed sea of azure, the Lesser Antilles stretch from Puerto Rico to Venezuela, their Windward Island chain forming the eastern barrier of the Caribbean Sea. Viewed from the air, ragged blots of green and brown erupt from the blue void, whose breakers fringe them with nervous fingers of foam that glisten white in the sun and wander aimlessly up and down the sandy beaches and reefs, or smear the rocky coastline with an inconstant icing of cotton candy in its upward leap toward volcanic spines and spires. Ta-da-a-a!

So much for the travelogue, which should have gotten you in the mood (or out of it, as the case may be). There are those who believe if you have seen one island, you have seen them all, or that at best they offer only very little inducement for a return engagement. That viewpoint has some merit, certainly. On the other hand, one could take the position of Sportin' Life that it is perpetual summertime, and the livin' is easy, which has a lot of appeal in the dead of winter. The Caribbean Islands (or more precisely, the Windward Islands) generally look better from a clean cruise ship, with time off for swimming on the islands' marvelous beaches, than they do close up. They are lovely, and some of them offer some spectacular scenery; they also offer a lot of squalor and abject poverty, which can be hard to ignore.

St. Lucia is a good example of both. The approach is magnificent, with the town of Soufriére, gateway to the volcano of the same name, guarded by the twin spires of the Gros et Petit Pitons, which beggar description. Both Castries, the capital, and Soufriére are quaint, and quite French; sanitation is quite poor. The trip by schooner from Castries to Soufriére town for a visit to Soufriére volcano is a delight, passing as it does under craggy cliffs interspersed with valleys that end in lovely white sand beaches (some black sand ones, too) lined by waving palm trees.

Once at the South Seas Plantation on Captiva Island, in Florida, I watched pelicans being fed—maybe a hundred of them. It was a real show as the feeder tossed fish among them one at a time; it was sort of like watching a free-for-all after a football game that has gone sour, which I have once or twice, only more so. It was more like what happened as we sailed into (and later out of) Soufriére harbor as little black boys and young men, clad only in the briefest of briefs, leaped into the water and swam out toward (later, with) the boat, emitting the universal cry of "Dollah! Dollah!" As some bills were folded and flung out amongst the pelicans—uh, swimmers—there was agile activity that stopped just short of mayhem. To their credit, once firm possession was established, it was not further contested.

The major crisis in the world of finance these days has been the slide of the dollar against the pound sterling, the deutschmark, and the yen. Apparently the folks in the islands (and I expect elsewhere, except possibly the UK, Germany, and Japan) have either not heard about it, or at least could not care less. Over the years since just at the end of World War II, I have traveled to various, and sometimes rather far-flung, outposts during both high and low dollar years; I have yet to hear, "Pound! Pound!" or "Yen! Yen!" or "Deutschmark! Deutschmark!" (let alone "Ruble, Ruble"). Then, as now, most of the world's inhabitants would at least as soon acquire dollars as their own currency. The citizens of Grenada are widely and loudly singing the praises of Mr. Reagan and his infusion of dollars, as well as his military aid. Though Grenada is a member of the British Commonwealth, they do not say the same for the Queen.

Meanwhile, in Trinidad and the other islands (as well as a lot of other places over the world) they are still drinkin' rum (which they make there better than anywhere else) an' Coca-Cola, (though more often now in punch—I think) and both mothah an' daugtah (not to mention papa) are workin' (directly or indirectly) for (in the sense of "to get") the Yankee dollah!

J.B.T.

our mail box

Obstetrical Services in Tennessee

To the Editor:

In reference to the report by Fredia Wadley, M.D., from the Department of Health and Environment in the February TMA *Journal* (*J Tenn Med Assoc* 80:94-95, 1987), I am sure she is aware this is a problem not only across the state but in the country.

It is noted that the medical reimbursement fee in Tennessee is $650 to $725. Is the higher fee for obstetricians? After noting that in rural communities family practitioners and GPs provide most of the OB care, it is unfortunate that they are discriminated against for providing the same service.

Also reimbursement for delivery and hospital care is only $325 when most of the risk for pregnancy is being taken during this period.

All of us know that patients who are on Medicaid are those of highest risk and more litigious than private pay patients. To entreat physicians to care for these patients, it may be that reimbursement at levels higher than present may be conducive at least toward paying higher malpractice premiums.

Shelbyville will also soon be another community in Tennessee that will be without obstetrical services if nothing is done by the county/state. Everyone expects the highest service and perfect outcomes but no one is willing to pay the price for it.

Frank Jayakody, M.D.
841 Union St., Suite J
Shelbyville, TN 37160

Reply

I am aware of the obstetrical situation in Shelbyville. Commissioner Word and I visited Dr. Donald Barnes before he deleted obstetrical services from his practice and have worked closely with the health department as they tried to find physicians to provide this care.

Dr. Jayakody's first concern was the differential in reimbursement for obstetrical care ($650 to $725). The higher fee is for obstetricians as he suspected. Obstetricians maintain that they have more high risk and complicated cases in their practice which deserves differential in payment. I would be the first to admit that it would be better to pay everyone the same fee for an uncomplicated pregnancy, labor and delivery, and then have higher reimbursement rates for various complications or added reimbursements for additional procedures needed with high risk patients. We are slowly moving in this direction, but we usually run out of funds before we run out of needs.

Another concern he expressed was that $325 for delivery and hospital care is not an appropriate share of the total reimbursement, when most of the risk of the pregnancy is being taken during this period. However, I am sure he recognizes that without the prenatal care the risk at delivery is greater. If Medicaid reimbursed three-fourths or more of the global fee just for delivery and hospital care, there would be less incentive to see these patients early and provide the necessary prenatal care. Early and continuous prenatal care has repeatedly been shown to improve the outcome of pregnancy.

His next point was that if Medicaid reimbursed at a higher rate, then physicians might be able to use this on their higher malpractice premiums and continue with obstetrical care. This is precisely what we thought in May 1986, when we increased the reimbursement rate for obstetrical care. I realize $650 to $725 is not the usual and customary fee for payments, but it is higher than the Medicaid rate is in most states and a rate that we were proud to offer physicians in Tennessee. The Medicaid program would truly like to pay physicians their usual fee for every service, but the funds will probably never be available to do this.

The malpractice issue is frustrating to everyone, and I can understand how private physicians feel when they try to provide necessary health services to indigent and Medicaid patients, deal with the many third party payors, and still do battle with increasing malpractice premiums.

I am sure Dr. Jayakody sometimes feels that no one is concerned about the problems private physicians are facing. This is really not the case. We also are frustrated, but do not have nearly enough answers to the indigent health care funding issue. But I want to add that we will always listen to physicians and make whatever changes we can.

Fredia S. Wadley, M.D.
Chief Medical Officer
Dept. of Health and Environment
Cordell Hull Bldg.
Nashville, TN 37219

Medical Practice—1987 Style

To the Editor:

I have become quite concerned as I have watched the practice of medicine unfold in this community, as well as nationwide. We have a profession that is in the greatest demand and yet we have voluntarily given up our freedom to Medicare, the HMOs, the PROs, the Health Nets, CareChoices, the Mid-South Foundation, etc. We have let anyone who wanted to dictate to us how we practice medicine and what we charge for it. The aforementioned groups have successfully made a very poorly disguised attempt and have been successful at reducing our income and, at the same time, enhancing their own. The money that comes out of your pocket goes to pay the middle man, who has never been in the picture before and who should not be there now.

The total cost of health care is not going down. The money is just being redistributed. Every one of you works as a physician and in medicine to make a living. There are not too many saints out there among you who would be practicing medicine if you were not making a living at it. So, therefore, I think we need to approach this problem from a different stance. And I think we need to play hard ball!

Higher medical organizations have relied on political lobbying, which with either party is a farce. We are living under one of the most oppressive governments in history, and we are going hat-in-hand to politicians saying "Please don't tighten the noose one more notch." We believe that, if they do tighten it, we'll just be able to adapt to it. The Jews in Nazi Germany thought that each step would be acceptable and that they could tolerate each and every change.

If we don't stand together in a strong union support group, or whatever you want to call it, our profession as we know it is done. We must be prepared to ignore the law, to withhold services, to do whatever it takes to maintain our own integrity and maintain control of our own destiny. Each of you, and I am including myself, has joined organizations that have felt free to lower our fees, to withhold payment, to delay payment, the idea being that we are going to get more patients. I don't know where you think you are going to get more patients from, unless it's to take them away from your fellows. We are so afraid of losing one patient or one dollar that we are willing to sacrifice our freedom for someone's guarantee that we will be supplied patients. We're fools! Medicare has directly intervened into the practice of ophthalmology and anesthesiology to my knowledge, as of this time; it has reduced fees by a minimum of 25% and is looking at 15% to 20% more. I know of no other business that can stand that type of reduction.

It doesn't take every physician in America to stand together and tell the organizers that we will not work for them. It would only take the physicians of Williamson County or Davidson County, or both. No one is going to help us except ourselves. No politician is going to help you to save your professional and financial integrity. In fact, they are working in the opposite direction. Your voice amounts to nothing. The only thing you have is your skill as a physician and your ability to say when you will work and who you will work for.

I think the hour is very late. How much will you take before you react?

John M. Omohundro, III, M.D.
1325 Carters Creek Pike
Franklin, TN 37064

in memoriam

William F. Encke, age 60. Died February 19, 1987. Graduate of Tulane University School of Medicine. Member of Williamson County Medical Society.

Shelby James Gregory, age 29. Died March 3, 1987. Graduate of University of Tennessee School of Medicine. Member of Carter County Medical Society.

Harry T. Moore, Jr., age 66. Died February 24, 1987. Graduate of University of Tennessee College of Medicine. Member of Nashville Academy of Medicine.

Brian T. Shorney, age 65. Died February 15, 1987. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

BLOUNT COUNTY MEDICAL SOCIETY
Ricky T. Mohon, M.D., Maryville
Melissa E. Trekell, M.D., Maryville

CARTER COUNTY MEDICAL SOCIETY
Robert E. Grindstaff, M.D., Johnson City

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY
Mark William Bookout, M.D., East Ridge
Biram C. Hicks, M.D., Chattanooga
Karim Naguib Iskander, M.D., Rossville, GA

CONSOLIDATED MEDICAL ASSEMBLY OF WEST TENNESSEE
Charles McCall, M.D., Jackson

KNOXVILLE ACADEMY OF MEDICINE
Terry Lee Adams, M.D., Knoxville
Wilson C. Beamer, M.D., Knoxville
Gregg H.D. Kesterson, M.D., Knoxville
Frederick A. Klein, M.D., Knoxville
Thomas J. Ryan, M.D., Knoxville
Hafeezul H. Siddiqui, M.D., Knoxville

MARSHALL COUNTY MEDICAL SOCIETY
George Bone, M.D., Lewisburg

MEMPHIS-SHELBY COUNTY MEDICAL SOCIETY
Gary Kimzey, M.D., Memphis

NASHVILLE ACADEMY OF MEDICINE
Yilmaz Eryasa, M.D., Nashville

NORTHWEST TENNESSEE ACADEMY OF MEDICINE
George Gasel, M.D., Union City
Ramakant M. Mulay, M.D., Dyersburg

WASHINGTON-UNICOI-JOHNSON COUNTY MEDICAL ASSOCIATION
Stephen T. Box, M.D., Johnson City
Sue McCoy, M.D., Johnson City
Carol F. Owais, M.D., Johnson City
Wisam N. Owais, M.D., Johnson City
T. Allen Polk, M.D., Piney Flats

WILLIAMSON COUNTY MEDICAL SOCIETY
William R. Halliday, M.D., Brentwood

personal news

Wayne T. Scott, M.D., Chattanooga, has been certified as a Diplomate of the American Board of Internal Medicine.

announcements

CALENDAR OF MEETINGS

NATIONAL

June 1-5	International Conference on AIDS (sponsored by US Public Health Service and World Health Organization)—Washington Hilton, Washington, D.C.
June 7-9	American Diabetes Association—Hyatt Regency, Indianapolis
June 10-12	Endocrine Society—Indianapolis
June 11-13	34th Annual Mountaintop Medical Assembly (sponsored by Haywood County [NC] Medical Society)—Waynesville Country Club, Waynesville, N.C.
June 11-14	American Association of Neuropathologists—Seattle
June 13-16	Eye Bank Association of America—Grand Hotel, Mackinac Island, Mich.
June 15-17	NIH Consensus Development Conference on the Management of Clinically Localized Prostate Cancer (cosponsored by National Cancer Institute)—Bethesda, Md.
June 26-27	Association of Pakistani Physicians—Chicago
June 28-July 2	American Orthopedic Society for Sports Medicine—Orlando, Fla.
July 3-5	North Carolina Medical Society, 17th Annual Sports Medicine Symposium—Blockade Runner Hotel, Wrightsville Beach, N.C.
July 5-9	Health Physics Society—Hotel Utah/Salt Lake City Convention Center
July 10-12	Olympic Festival Congress on Sports Medicine and Science—Part I: Sports Medicine and Science Issues and Trends—Univ. of North Carolina, Chapel Hill
July 13-15	Olympic Festival Congress on Sports Medicine and Science—Part II: Athletic Injuries Prevention and Treatment—Duke Univ., Durham, N.C.
July 13-15	NIH Consensus Development Conference on Neurofibromatosis—Bethesda, Md.
July 21-26	World Association of Societies of Pathology (Anatomic and Clinical)—Sheraton Hotel, Washington, D.C.
July 29-Aug. 3	International Doctors in Alcoholics Anonymous—Lexington Marriott Resort, Lexington, Ky.

TMA Members Receive AMA Physician's Recognition Award

Twenty-nine TMA members qualified for the AMA Physician's Recognition Award during February 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Maysoon S. Ali, M.D., Waverly
Subhi D. S. Ali, M.D., Waverly
Ira L. Arnold, M.D., Chattanooga
Leland L. Atkins, M.D., Memphis
George F. Bale, M.D., Memphis
Robert R. Bendt, M.D., Nashville
Terry M. Bingham, M.D., Harriman
Donald R. Campbell, M.D., Chattanooga
Nicholas H. Edwards, M.D., Grand Junction
William E. Elliott, M.D., Maryville
John W. Ellis, M.D., Trenton
Charles W. Hawkins, M.D., Chattanooga
John W. Lamb, M.D., Nashville
Stephen M. Lazarus, M.D., Knoxville
Vichien Lorch, M.D., Knoxville
Herbert T. McCall, M.D., Madison
Oscar M. McCallum, M.D., Henderson
Alexander C. McLeod, M.D., Nashville
O. L. Merritt, M.D., Dandridge
James B. Millis, M.D., Nashville
James N. Moore, M.D., Nashville
Lovett P. Reddick, M.D., Kingsport
Allen L. Schlamp, M.D., Jackson
Abram C. Shmerling, M.D., Nashville
Avron A. Slutsky, M.D., Memphis
Robert C. Taylor, M.D., Signal Mountain
Susan G. Thurmond, M.D., Memphis
William P. Titus, III, M.D., Nashville
George H. Wood, M.D., Knoxville

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Abstract of the Proceedings of the House of Delegates Of the Tennessee Medical Association Knoxville, Tennessee—April 8-11, 1987

Call to Order

The 152nd annual meeting of the Tennessee Medical Association was conducted in Knoxville, Tennessee, April 8-11, 1987, with headquarters in the Hyatt Regency Hotel. The House of Delegates met initially at 3:00 PM, April 8, 1987, with Malcolm R. Lewis, M.D., Nashville, presiding as speaker of the House and F. Hammond Cole, Jr., M.D., Memphis, as vice speaker.

Invocation

At the opening session, John H. Burkhart, M.D., Knoxville, gave the invocation: "Let us pray. Almighty God, when we consider the heavens, the work of your hands, the earth, the moon and the stars which you have ordained, what is man that you are mindful of him? And yet you have created him and you have given him life and health and joy. But you have also decreed the inevitability of his death, the threats to his health and the uncertainty of his pleasure. We believe that to better protect him against those forces which assail him, you have called to be your servants some who, by motivation, training and proper behavior, have the honor to be called physicians. And that is why we are here today. To meet together, to work and to plan how to humbly deserve the title physician and how to faithfully respond to your charge. Bless us in our efforts, for this is our constant need. Amen."

Report of the Committee on Credentials

Mary B. Duffy, M.D., Knoxville, chairman of the Committee on Credentials, reported there was a quorum present. The speaker declared the House was in session.

1986 Minutes Approved

The speaker announced that an abstract of the minutes of the last regular session of the House of Delegates was reproduced in the June 1986 issue of the *Journal of the Tennessee Medical Association*. It was moved and seconded that the abstracted minutes of the 1986 session of the House of Delegates be approved as published in the June 1986 issue of the *Journal*. The motion was adopted.

Reference Committees

The speaker announced the members of the reference committees to consider reports, resolutions, amendments, and all matters requiring action by the House of Delegates.

REFERENCE COMMITTEE ON CREDENTIALS
Mary B. Duffy, M.D., Knoxville, *Chairman*
Hugh E. Green, M.D., Carthage
Eugene J. Spiotta, Jr., M.D., Memphis

REFERENCE COMMITTEE ON AMENDMENTS TO THE CONSTITUTION AND BYLAWS
John H. Burkhart, M.D., Knoxville, *Chairman*
John L. Farringer, Jr., M.D., Nashville
C. Eugene Jabbour, M.D., Memphis

REFERENCE COMMITTEE A
H. Victor Braren, M.D., Nashville, *Chairman*
Dennis A. Higdon, M.D., Memphis
Marvin D. Peterson, M.D., Maryville

REFERENCE COMMITTEE B
Oscar M. McCallum, M.D., Henderson, *Chairman*
Hal S. Stubbs, M.D., Bristol
Cullen R. Merritt, II, M.D., Nashville

REFERENCE COMMITTEE C
Thomas A. Currey, M.D., Memphis, *Chairman*
Lawrence R. Nickell, M.D., Columbia
R. Leslie Hargrove, M.D., Knoxville

REFERENCE COMMITTEE D
Richard M. Pearson, M.D., Memphis, *Chairman*
Randall G. Samples, M.D., Cookeville
Joel E. Avery, M.D., Chattanooga

COMMITTEE ON OUTSTANDING PHYSICIAN OF THE YEAR AWARD
Nat E. Hyder, Jr., M.D., Johnson City, *Chairman*
Thomas K. Ballard, M.D., Jackson
Clarence R. Sanders, M.D., Gallatin

Nominating Committees

As required in the Bylaws, the Board of Trustees appointed a Nominating Committee with representatives from each of the three grand divisions of the state. The speaker announced the committee members.

EAST TENNESSEE

Duane C. Budd, M.D., Johnson City
Pete S. Soteres, M.D., Chattanooga
R. Kent Farris, M.D., Knoxville

MIDDLE TENNESSEE

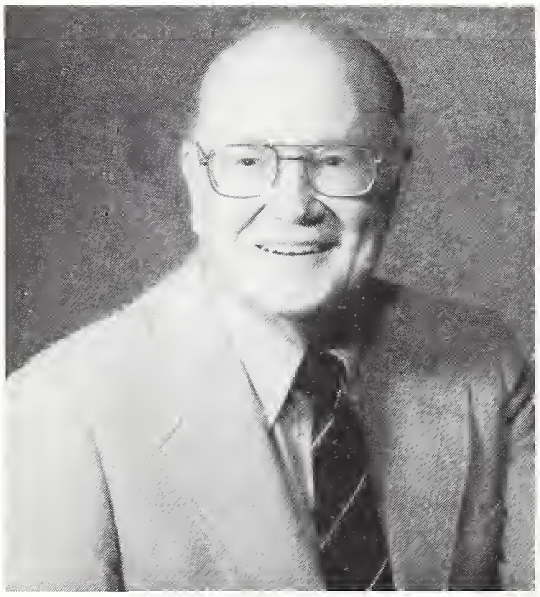
William J. Pedigo, Jr., M.D., Clarksville
James W. Hays, M.D., Nashville
Charles E. Jordan, III, M.D., Cookeville

WEST TENNESSEE

James W. Shore, M.D., Martin
Phillip A. Pedigo, M.D., Memphis
Charles W. White, M.D., Lexington

ELECTION BY HOUSE OF DELEGATES APRIL 11, 1987

The preliminary report of the Nominating Committee was presented in the first session of the House of Delegates on Wednesday, April 8, 1987. The final report of the Nominating Committee was presented on Saturday, April 11, 1987 at the closing session of the House. Nominees submitted by the committee were voted upon individually, and in each instance the speaker called for additional nominations from the floor. The following were elected.



Newly elected President-Elect
John B. Thomison, M.D., Nashville

President-Elect—John B. Thomison, M.D., Nashville
Speaker—F. Hammond Cole, Jr., M.D., Memphis
Vice-Speaker—George H. Wood, M.D., Knoxville
Vice-President (West Tennessee)
Thomas A. Currey, M.D., Memphis
Vice-President (Middle Tennessee)
William C. Beazley, M.D., Clarksville
Vice-President (East Tennessee)
Clarence Goulding, Jr., M.D., Johnson City
AMA Delegate (Middle Tennessee)
John B. Thomison, M.D., Nashville
(January 1, 1988-December 31, 1989)
AMA Alternate Delegate (Middle Tennessee)
Clarence R. Sanders, M.D., Gallatin
(January 1, 1988-December 31, 1989)
AMA Alternate Delegate (Middle Tennessee)
Clarence R. Sanders, M.D., Gallatin
(April 11, 1987-December 31, 1987)—filling unexpired term of Dr. George W. Holcomb, Jr.
AMA Delegate (Middle Tennessee)
John S. Derryberry, M.D., Shelbyville
(January 1, 1988-December 31, 1989)
AMA Alternate Delegate (Middle Tennessee)
Thurman L. Pedigo, M.D., McMinnville
(January 1, 1988-December 31, 1989)
AMA Delegate (West Tennessee)
Allen S. Edmonson, M.D., Memphis
(January 1, 1988-December 31, 1989)
AMA Alternate Delegate (West Tennessee)
Hugh Francis, Jr., M.D., Memphis
(January 1, 1988-December 31, 1989)
AMA Alternate Delegate (West Tennessee)
A. Roy Tyrer, Jr., M.D., Memphis
(January 1, 1988-December 31, 1988)—filling unexpired term of Dr. Allen S. Edmonson
AMA Delegate (State-at-Large)
George A. Zirkle, Jr., M.D., Knoxville
(April 11, 1987-December 31, 1988)
AMA Alternate Delegate (State-at-Large)
Francis W. Gluck, Jr., M.D., Nashville
(April 11, 1987-December 31, 1988)
AMA Alternate Delegate (State-at-Large)
James R. Royal, M.D., Chattanooga
(April 11, 1987-December 31, 1988)—filling unexpired term of Dr. George A. Zirkle, Jr.
AMA Young Physician Section Delegate
James C. Fleming, M.D., Memphis (1987)
AMA Young Physician Section Alternate Delegate
Ann H. Price, M.D., Nashville (1987)

TRUSTEES

East Tennessee:
Hal S. Stubbs, M.D., Bristol (1990)

Middle Tennessee:
William M. Young, M.D., Fayetteville (1990)

West Tennessee:
Evelyn B. Ogle, M.D., Memphis (1990)
James T. Craig, Jr., M.D., Jackson (1990)

COUNCILORS

Second District—Robert M. Overholt, M.D., Knoxville (1989)

Third District—John F. Boxell, M.D., Chattanooga (1988)—filling unexpired term of Dr. Robert W. Myers, Sr.

Fourth District—Will G. Quarles, Jr., M.D., Livingston (1989)

Sixth District—Howard L. Salyer, M.D., Nashville (1989)

Eighth District—Montie E. Smith, Jr., M.D., Selmer (1989)

Tenth District—Dennis A. Higdon, M.D., Memphis (1989)

THE ABOVE WERE ELECTED BY THE HOUSE OF DELEGATES

AMENDMENTS TO THE CONSTITUTION AND BYLAWS

The speaker reported that there was one amendment to the Constitution lying on the table, one new amendment to the Constitution, and two new amendments to the Bylaws to be considered at this session by the House.

The proposed amendments to the Constitution and Bylaws are shown below, with proposed new language shown in **boldface** type and material to be deleted shown in *italics* and enclosed in brackets.

AMENDMENTS TO THE CONSTITUTION LYING ON THE TABLE

CONSTITUTIONAL AMENDMENT NO. 1-86

Dues Exemption for Retired Members

Whereas, Physicians who have retired from the active practice of medicine before the age of 65 may be financially unable to pay the required dues; and

Whereas, The physicians may have been members of organized medicine for a number of years; and

Whereas, These physicians are still important members of the medical community. Now, therefore be it

RESOLVED, That Article IV of the Constitution be amended as follows:

Sec. 4. Veteran members are those who, because of age, [*or*] impaired health, **or retirement,** have been elected veteran members of their component societies, and who are so certified to the Association annually by the component societies. Members who have attained age 70 **or have fully retired** in the previous calendar year [*, or age 65 if fully retired,*] may be elected veteran members for the current and subsequent years **provided they have been members in good standing for the previous five years.** Members who are not practicing medicine because of impaired health may be elected veteran members during the first year of disability and thereafter for as long as the disability exists.

ACTION: **NOT ADOPTED**

AMENDMENTS TO THE CONSTITUTION

CONSTITUTIONAL AMENDMENT NO. 1-87

Medical Student Section

Whereas, The House of Delegates of the Tennessee Medical Association has voted to establish a Medical Student Section. Now, therefore be it

RESOLVED, That Article VI of the Constitution be amended by adding a new Section 3 as follows:

Sec. 3. There shall be a Medical Student Section to provide representation for the interests of medical students within the structure of the Association. The medical students of each Liaison Committee on Medical Education accredited medical school in the state shall be entitled to representation in the section. All members must be members of the Association. The Medical Student Section shall be organized under a governing body and shall elect one delegate to represent it in the House of Delelgates of the Association.

ACTION: **Lying on the table to be acted upon during the House of Delegates session at the 1988 annual meeting.**

AMENDMENTS TO THE BYLAWS

BYLAW AMENDMENT NO. 1-87

Committee on Group Insurance

Whereas, The Tennessee Medical Association Board of Trustees, in July of 1985, authorized the formation of The TMA Association Insurance Agency, Inc. for the purpose of consolidating the various TMA sponsored group insurance programs into one agency with one administrator; and

Whereas, The TMA Association Insurance Agency, Inc. now provides a comprehensive and broad range of improved insurance programs for TMA members, families, and employees; and

Whereas, The TMA Association Insurance Agency, Inc. is managed by experienced, competent insurance professionals with appropriate physician input, including its president, who is the former chairman of the TMA Insurance Committee; and

Whereas, This agency is serving the Association well and there is no longer a need for a committee on group insurance within the Association's organizational structure. Now, therefore be it

RESOLVED, That Chapter VII, Section 12 of the Bylaws be amended by deletion and the following sections renumbered accordingly:

[*Sec. 12. The Committee on Tennessee Medical Association Group Insurance shall be composed of not more than six members representing the three grand divisions of the state. It shall be the duty of this committee to attend to all group insurance in which this Association is or may become interested. It shall have the power to select insuring companies, accept or reject master policies, arrange premium rates, and act as trustees for this Association in the matter of such group insurance. All actions of the committee shall be subject to the approval of the Board of Trustees. The chairman of the committee shall be designated by the Board of Trustees. He shall report annually to the House of Delegates.*]

REFERENCE COMMITTEE ON AMENDMENTS TO THE CONSTITUTION AND BYLAWS—*recommended adoption of Bylaw Amendment No. 1-87.*

ACTION: **ADOPTED**

BYLAW AMENDMENT NO. 2-87

Nominating Committee

RESOLVED, That Chapter IV of the Bylaws be amended as follows:

Sec. 2. On or before March 1 each year, preceding the annual meeting, the Board of Trustees shall select nine delegates to compose a Nominating Committee. **The Board of Trustees shall also appoint a non-voting advisory member from the Committee on Constitution and Bylaws.** . . .

REFERENCE COMMITTEE ON AMENDMENTS TO THE CONSTITUTION AND BYLAWS—*recommended adoption of Bylaw Amendment No. 2-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTIONS

The reference committees have the option of recommending a resolution for adoption or rejection, for adoption as amended or substituted, for referral, or for no action. The resolutions that follow are in the form in which they were **adopted, not adopted,** or **referred** by the House of Delegates. Resolutions No. 6-87 and No. 23-87 were withdrawn.

RESOLUTION NO. 1-87

Sunset Provision for Resolutions

BY: ARDEN J. BUTLER, JR., M.D., CHAIRMAN
TMA BOARD OF TRUSTEES

Whereas, In 1980, the House of Delegates adopted policy calling for resolutions to automatically become void after seven years, unless reintroduced and reaffirmed by the House of Delegates; and

Whereas, This procedure has been extremely effective in the timely retirement of resolutions that have either outlived their purpose or action called for has been accomplished; and

Whereas, It is the desire of this House of Delegates to continue this sunset policy. Now, therefore be it

RESOLVED, That the House of Delegates reaffirm its policy for resolutions to expire after seven years, unless reintroduced and reaffirmed prior to that time; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE C—*recommended adoption of Resolution No. 1-87.*

ACTION: **ADOPTED**

RESOLUTION NO. 2-87

Mandatory Auto Safety Belt Usage

BY: ARDEN J. BUTLER, JR., M.D., CHAIRMAN
TMA BOARD OF TRUSTEES

Whereas, The General Assembly of the state of Tennessee enacted mandatory auto seat belt legislation which was codified in TCA 55-9-603; and

Whereas, Included in that statute is a provision for exemption based upon medical conditions; and

Whereas, To qualify under this exemption, the patient must be duly certified in writing by a physician as to the "nature of the handicap as well as the reason such restraint is inappropriate"; and

Whereas, Most authorities in the area of automobile safety indicate that exemptions based on medical conditions should be minimized. Now, therefore be it

RESOLVED, That the House of Delegates of the Tennessee Medical Association encourage the members of the Association to authorize exemption only in those instances where safety considerations afforded by seat belt usage are outweighed by the appropriateness of restraints given physical handicap; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE B—*recommended adoption of Resolution No. 2-87.*

ACTION: **ADOPTED**

RESOLUTION NO. 3-87

Reaffirmation of Peer Review Guidelines

BY: ARDEN J. BUTLER, JR., M.D., CHAIRMAN
TMA BOARD OF TRUSTEES

Whereas, In 1980, the Tennessee Medical Association adopted guidelines for peer review and mediation committees and recommended component societies also adopt such; and

Whereas, The Tennessee Medical Association in 1984 combined peer review and mediation functions into the Peer Review Committee; and

Whereas, The policy adopted in 1980 has worked well and should be continued. Now, therefore be it

RESOLVED, That the Tennessee Medical Association House of Delegates reaffirm previous policy and direct that future requests for third-party payors for claims review by the Tennessee Medical Association Peer Review Committee be denied in such cases when the carrier has made a prejudgment of the appropriateness of services rendered or the charges made for such services and has so informed the patient (policyholder) of that decision; and be it further

RESOLVED, That the Tennessee Medical Association component societies be informed of this action and recommend that the societies also adopt such a resolution refusing to honor requests for peer review when inflammatory statements have been made by the third-party payor to the patient; and be it further

RESOLVED, That the House of Delegates adopt as policy the concept that the Tennessee Medical Association Peer Review Committee be available to the patient for mediation of a complaint regarding treatment upon written request to the committee from the patient; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE C—*recommended adoption of Resolution No. 3-87.*

ACTION: **ADOPTED**

RESOLUTION NO. 4-87

Opposition to Fee Splitting

BY: HOWARD L. SALYER, M.D., CHAIRMAN
TMA JUDICIAL COUNCIL

Whereas, Medicine is an honorable profession with traditions and values that must be maintained in order to ensure the public's respect and trust; and

Whereas; It is in the best interest of the patient that referrals be primarily based upon competence of the physician to whom the patient is referred; and

Whereas, The splitting of fees creates an economic bias against such ethical referral principles; and

Whereas, The division of fees, or agreeing to divide or split a fee for professional services for referring a patient, is grounds for suspension or revocation of license to practice medicine according to Tennessee Code Annotated, Section 63-1-120 (12); and

Whereas, Fee splitting is also unethical according to the Current Opinions of the AMA Council on Ethical and Judicial Affairs 1986, Section 6.04; and

Whereas, A few physicians, who violate the code of ethics, may sully the honor of the entire profession. Now, therefore be it

RESOLVED, That the Tennessee Medical Association endorse the strict enforcement of Tennessee Code Annotated, Section 63-1-120 (12), and send a copy of this resolution to the commissioner of Health and Environment, asking for his support; and be it further

RESOLVED, That the membership of the Tennessee Medical Association be reminded of the unethical and illegal practice of fee splitting; and be it further

RESOLVED, That the membership of the Tennessee Medical Association become more familiar with the reports of the AMA Council on Ethical and Judicial Affairs; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE C—*recommended adoption of Resolution No. 4-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 5-87

Potentially Unethical Contracts

BY: HOWARD L. SALYER, M.D., CHAIRMAN
TMA JUDICIAL COUNCIL

Whereas, Physicians are faced with signing participation contracts from various HMOs, PPOs, and IPAs; and

Whereas, Most contracts are drawn to the benefit of the organization and not the physician; and

Whereas, Financial incentives are placed before the physician which do not always consider the physician's ethical responsibility to his patients; and

Whereas, Many contracts are being signed by physicians, which place them in potentially unethical situations by rewarding them for not referring patients. Now, therefore be it

RESOLVED, That the Tennessee Medical Association, through its communications, admonish physicians not to sign contracts that place them in potentially unethical situations by rewarding them financially for referring or not referring patients; and be it further

RESOLVED, That physicians be reminded they are the patients' advocates and have affirmative, ethical obligations to disclose to patients or referring colleagues their ownership-interest in a facility or therapy prior to utilization; and be it further

RESOLVED, That physicians are unethical when they exploit the patient in any way by inappropriate or unnecessary utilization; and be it further

RESOLVED, That when a physician's commercial interest conflicts so greatly with his patient's interest as to be incompatible, the physician should make alternative arrangements for the care of the patient; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE C—*recommended adoption of Resolution No. 5-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 7-87

Opposition to Two-Level Nursing Concept

BY: ARDEN J. BUTLER, JR., M.D., CHAIRMAN
TMA BOARD OF TRUSTEES

Whereas, Nursing education is of vital importance to the medical profession; and

Whereas, There are proposals nationally and by the Tennessee Nurses' Association calling for entry into the field of nursing to be at two levels, baccalaureate and associate degrees, instead of the present four levels, baccalaureate degree, associate degree, diploma, and the licensed practical nursing program; and

Whereas, There continues to be a shortage of nurses throughout Tennessee, and the two-level concept would further decrease the availability of nurses to hospitals and thereby increase the cost of medical care; and

Whereas, There is serious question as to whether there exists an adequate number of positions in baccalaureate-level nursing programs, as well as the availability of qualified instructors to meet the increased demand as a result of the implementation of the two-level proposal; and

Whereas, Former Governor Alexander created a Commission on Nursing to study this issue; and

Whereas, The Tennessee Medical Association, in accordance with the position previously adopted by the House of Delegates, provided testimony to the Commission on Nursing in opposition to the two-level concept of nursing education. Now, therefore be it

RESOLVED, That the Tennessee Medical Association continue to oppose the two-level concept of nursing and encourage expanded nursing training on all four levels, baccalaureate degree, associate degree, diploma, and the licensed practical nursing program; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE D—*recommended adoption of Resolution No. 7-87.*

ACTION: **ADOPTED**

SUBSTITUTE RESOLUTION NO. 8-87

Committee to Monitor Peer Review Organizations (PROs)

By: Arden J. Butler, Jr., M.D., Chairman
TMA Board of Trustees

Whereas, The Tennessee Medical Association has endorsed Mid-South Foundation for Medical Care as the Peer Review Organization (PRO) for Tennessee since 1983; and

Whereas, Physicians are patient advocates in matters relating to the function of the PRO; and

Whereas, The PRO needs more physician input to ensure the physician-patient relationship is maintained; and

Whereas, Recent activities and policy implementation by the PRO relating to retrospective denials and/or sanctions is potentially damaging to the physician and critical to quality patient care. Now, therefore be it

RESOLVED, That the Tennessee Medical Association House of Delegates instruct the Board of Trustees to establish an oversight committee to monitor the activities of the Peer Review Organization (PRO). This committee should be strongly supported by the staff to monitor the operations of the PRO and lend aid to Tennessee Medical Association members in appealing PRO sanctions; and be it further

RESOLVED, That the Tennessee Medical Association address those aspects of PRO sanctions which result in making the attending physician liable for minimally significant, unforeseen events; and be it further

RESOLVED, That physicians be encouraged to tape record conversations with the PRO for their own protection; and be it further

RESOLVED, That activity regarding retroactive denials and sanctions be voluntarily reported by physicians to the Tennessee Medical Association to facilitate the monitoring of the PRO; and be it further

RESOLVED, That a similar resolution be presented to the American Medical Association by the Tennessee delegation, with copies of the resolution going to the Tennessee congressional delegation, the director of the Health Care Financing Administration, and the Board of the Mid-South Foundation for Medical Care, Inc.; and be it further

RESOLVED, That the Tennessee Medical Association committee to monitor PRO report its findings directly to the American Medical Association Ad Hoc Committee on PRO in order to facilitate the dissemination of such information to all physicians; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*offered Substitute Resolution No. 8-87 to replace original Resolutions No. 8-87 and No. 17-87; recommended adoption of Substitute Resolution No. 8-87.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 9-87

Opposition to Physician Diagnosis Related Groups (DRGs)

By: Donald L. Gaines, M.D., Delegate
TMA Hospital Medical Staff Section

Whereas, The Office of Management and Budget has formulated a series of recommendations regarding the Medicare program; and

Whereas, One recommendation would combine payment for both physician services and hospital services for all hospital inpatient stays into one Diagnosis Related Group (DRG) payment; and

Whereas, The OMB proposal would also provide for a differential in reimbursement levels for participating and nonparticipating physicians; and

Whereas, Such a plan would be the forerunner for future efforts to provide services under a capitation system. Now, therefore be it

RESOLVED, That physicians must remain the patients' advocates in the patients' relationship with the hospital; and be it further

RESOLVED, That physicians should never compromise the quality of care they provide their patients in order to conserve costs; and be it further

RESOLVED, That physicians are entitled to compensation commensurate with services rendered and should not receive income for withholding appropriate care; and be it further

RESOLVED, That physicians' reimbursements should not be included with hospital reimbursement in a global fee; and be it further

RESOLVED, That any physician economic incentive plan should be reviewed by the medical staff with assistance of independent legal counsel; and be it further

RESOLVED, That the Tennessee delegates to the American Medical Association House of Delegates introduce a similar resolution; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*recommended adoption of Resolution No. 9-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 10-87

Disability Evaluation

By: Duane C. Budd, M.D., Chairman
TMA Medical Practice Committee

Whereas, Many patients in the state of Tennessee are truly disabled and need financial aid; and

Whereas, Disability decisions in Tennessee are made by the Disability Determination Section (DDS), a division of the Department of Human Services; and

Whereas, A recent court decision in a class action suit, if properly implemented, will result in a fundamental change in the disability determination process by requiring the DDS to: (1) give greater weight to the opinion of the treating physician than to the opinions of hired consultants, (2) specifically ask the treating physician to assess an individual's ability to perform work-related activities, and (3) obtain necessary evaluations and testing from treating physicians rather than hired consultants in all cases where the treating physician is willing and qualified to perform the services; and

Whereas, The Tennessee Medical Association believes that the above changes would be beneficial. Now, therefore be it

RESOLVED, That the Tennessee Medical Association encourage cooperation by the membership with the Disability Determination Section whereby the treating physician may have greater input into the disability determination of the individual; and be it further

RESOLVED, That physicians be encouraged to report to the Tennessee Medical Association instances of noncompliance with the directives of the court; and be it further

RESOLVED, That a copy of this resolution be sent to the Disability Determination Section of the Department of Human Services; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE B—*recommended adoption of Resolution No. 10-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 11-87

HIV Infection and AIDS

By: Arden J. Butler, Jr., M.D., Chairman
TMA Board of Trustees

Whereas, The conditions related to the human immunodeficiency virus (HIV) are manifested in a significant portion of the population through acquired immunodeficiency syndrome (AIDS) and other manifestations; and

Whereas, HIV is a retrovirus whose genome becomes permanently integrated into its host's genetic material, and thus may infect any human host for life; and

Whereas, As many as 1.5 to 2.0 million people in the United States may be infected with HIV, and an undetermined but significant portion of those will develop AIDS; and

Whereas, By early 1987 over 30,000 of all HIV infected men, women and children from all states in the United States, and over 175 in Tennessee, had been diagnosed with AIDS; and

Whereas, The potential medical, social, legal, psychological and economic impact of HIV infection (including AIDS) on the people of Tennessee within the next ten years represents a large and unavoidable burden on health care resources; and

Whereas, Any person infected by HIV can potentially transmit the virus to others; and

Whereas, Transmission of HIV occcurs as a result of sexual intercourse, a sharing of blood, or from infected mothers to their infants, and there is no known risk of nonsexual infection in most of the situations people encounter in their daily lives; and

Whereas, There is presently no vaccine to prevent HIV infection and no cure for AIDS; and

Whereas, The most effective deterrent to preventing AIDS is the prevention of HIV transmission to unexposed people, and aggressive public health education may be the most effective way to reduce the risk of HIV infection; and

Whereas, The occurrence of HIV infection can be prevented by sexual practices which prevent the transmission of HIV; and

Whereas, Physicians are uniquely qualified to care for and counsel HIV infected patients and are looked upon by the community as resources for information and education about HIV and AIDS. Now, therefore be it

RESOLVED, That the Tennessee Medical Association urge all physicians and other health care providers in Tennessee to provide appropriate care to human immunodeficiency virus (HIV) infected individuals and to those with acquired immunodeficiency syndrome (AIDS); and be it further

RESOLVED, That the Tennessee Medical Association establish a committee on HIV infection (including AIDS) for the purpose of serving the Tennessee Medical Association, allied health care professionals, government officials and others in Tennessee as a primary resource for information on the diagnosis, prevention and treatment of HIV infection (including AIDS) and on the care of patients with HIV infection (including AIDS); and be it further

RESOLVED, That the Tennessee Medical Association consider a continuing medical education program for physicians in Tennessee on the immunovirological, clinical, epidemiologic and psychosocial aspects of HIV infection (including AIDS); and be it further

RESOLVED, That the Tennessee Medical Association immediately provide the necessary resources to encourage the commissioner of Health and Environment to establish a task force on HIV infection (including AIDS) composed of a broad cross-section of health care professionals, as well as representatives of state and local governments, public and private education, law, the clergy and other groups whose fundamental purposes include the prevention of HIV infection (including AIDS) and the treatment of patients with HIV infection (including AIDS); and be it further

RESOLVED, That the Tennessee Medical Association, in coordination with component medical societies and medical specialty societies in Tennessee, make available to all health care professionals in Tennessee recommendations on providing for the needs of those infected with HIV (including AIDS); and be it further

RESOLVED, That the Tennessee Medical Association support legislative proposals and public policy related to HIV infection (including AIDS) that adequately consider the scientific evidence on the occurrence and transmission of the HIV virus; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE B—*recommended adoption of Resolution No. 11-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

SUBSTITUTE RESOLUTION NO. 12-87

PROs: Medical Necessity Denial, Retrospective Review, and Physician Responsibilities

By: Phil E. Orpet, Jr., M.D.
Tennessee Society of Internal Medicine
Memphis-Shelby County Medical Society

Whereas, The Mid-South Foundation for Medical Care, Inc., which serves as the Peer Review Organization (PRO) in Tennessee, is required to complete medical necessity review in 33 days from the receipt of records from the hospital to the mailing of notice of medical necessity *denial* to the patient; and

Whereas, This rapid time requirement has resulted in denials before the involved physician has had ample opportunity to add information to the review that would establish medical necessity; and

Whereas, These denials are then considered as "technical denials" (on a time basis) rather than "legitimate denials" (on a medical review basis); and

Whereas, Many patients receive letters of denial for procedures as being medically unnecessary, and most of those, on reconsideration, are approved. Now, therefore be it

RESOLVED, That the Tennessee delegates to the American Medical Association House of Delegates submit a resolution requesting that the American Medical Association strongly urge the Health Care Financing Administration (HCFA) to revise the constrictive time frame for medical necessity review by the Peer Review Organizations (PROs) and to stop sending letters of denial to patients for procedures as being medically unnecessary until after reconsideration of each case; and be it further

RESOLVED, That the Tennessee Medical Association offer input to the PRO in attempts to modify the retrospective regulations as they stand now, recognizing that a review organization composed of our peers is far preferable to one composed of non-physicians; and be it further

RESOLVED, That Tennessee Medical Association members be made aware of the fact that the American Medical Association has recommended to the HCFA the institution of a formal rule-making process for developing a procedure for the handling of second surgical opinions and quality care denials under the PRO mechanism; and be it further

RESOLVED, That a copy of this resolution be forwarded to the American Medical Association, the Tennessee congressional delegation, HCFA, and Mid-South Foundation for Medical Care, Inc.; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*offered Substitute Resolution No. 12-87 to replace original Resolutions No. 12-87 and No. 20-87; recommended adoption of Substitute Resolution No. 12-87.*

ACTION: **ADOPTED**

SUBSTITUTE RESOLUTION NO. 13-87

Drug Prescribing Environment

By: Phil E. Orpet, Jr., M.D.
Tennessee Society of Internal Medicine

Whereas, The prescription of a drug represents the culmination of a deliberative process between physician and patient, aimed at the prevention, amelioration or elimination of a disease or disorder; and

Whereas, The advocacy of a drug is a vital aspect of this process; and

Whereas, Many Tennessee pharmacists are apparently unaware of the requirement to substitute only generics from the approved Medicaid list when physicians sign the prescription "May Substitute" or give the brand name drug if there is no approved generic substitute; and

Whereas, The physicians of Tennessee must be concerned about their control and liability in prescribing drugs in the current confused environment characterized by patent drugs, brand name (formerly patented) drugs, competitive generic equivalents, mandated mail order drug prescription plans, bizarre drug pricing practices, and confused information regarding bioequivalents and bioavailability of brand name drugs vs generics. Now, therefore be it

RESOLVED, That the Tennessee Medical Association seek to sponsor a conference for interested participants to study the current environment of drug prescribing and pricing in Tennessee; and be it further

RESOLVED, That the agenda of such a conference include discussions of patented drugs, brand name drugs, competitive generic equivalents, mandated and other mail order prescription plans, data concerning bioavailability and bioequivalents of generic vs brand name drugs, Medicaid requirements, Tennessee law in regard to drug prescribing, medical liability, and other factors contributing to the present confusing environment; and be it further

RESOLVED, That the Tennessee Medical Association disseminate information and advice regarding prescribing of drugs generated by this conference to the membership via the *Journal of the Tennessee Medical Association* and to appropriate Tennessee Medical Association committees if any action is recommended by the conference; and be it further

RESOLVED, That a similar resolution be submitted to the American Medical Association House of Delegates by the Tennessee delegation; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE D—*offered Substitute Resolution No. 13-87 in place of original Resolution No. 13-87; recommended adoption of Substitute Resolution No. 13-87.*

ACTION: **ADOPTED**

SUBSTITUTE RESOLUTION NO. 14-87

Formation of a TMA Medical Student Section

By: Kent Kyger, M.D.
Nashville Academy of Medicine

Whereas, Medical students in Tennessee have a genuine interest in the activities of organized medicine within this state and the nation; and

Whereas, The American Medical Association has a policy that active participation by students is vital to the future of the Federation and the unity of the medical profession; and

Whereas, Since the American Medical Association formed a Medical Student Section in 1972, student participation in organized medicine has grown, with a current student AMA membership of over 30,000 with student representatives in the House of Delegates, on the Board of Trustees, and on the AMA Councils; and

Whereas, The medical student sections of many state medical associations have actively contributed in policy-making processes and community programming, providing an additional resource for both the state medical associations and the medical students. Now, therefore be it

RESOLVED, That the Tennessee Medical Association form a Medical Student Section to provide a forum within the Tennessee Medical Association for the exchange of information among students in medical school and their more senior colleagues; and be it further

RESOLVED, That the Tennessee Medical Association include medical students in the decision and policy-making process affecting all physicians in the state of Tennessee; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE D—*offered Substitute Resolution No. 14-87 to replace original Resolutions No. 14-87 and No. 25-87; recommended adoption of Substitute Resolution No. 14-87.*

ACTION: **ADOPTED**

RESOLUTION NO. 15-87

Ban on Sale and Use of Tobacco in Hospitals

By: Kent Kyger, M.D.
Nashville Academy of Medicine

Whereas, In 1980, the Tennessee Medical Association House of Delegates adopted a resolution deploring the indiscriminate use and sale of tobacco in hospitals; and

Whereas, Evidence continues to mount that the use of tobacco is associated with many health problems, notably cancer of the lung, emphysema, heart trouble, cancer of the pancreas, cancer of the bladder, and other diseases; and

Whereas, Physicians as community leaders should take a lead role in educating their patients regarding the harmful effects of tobacco and encourage them to quit smoking. Now, therefore be it

RESOLVED, That the Tennessee Medical Association, through its component societies, urge all hospitals in the state to ban the sale and use of tobacco in hospitals; and be it further

RESOLVED, That the Tennessee Hospital Association be encouraged to pass a similar resolution; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE B—*recommended adoption of Resolution No. 15-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 16-87

Medical Liability Insurance as a Prerequisite for Medical Staff Appointment

By: C. Eugene Jabbour, M.D.
Memphis-Shelby County Medical Society

Whereas, In the past, limits of medical liability insurance were not a requirement for medical staff appointment; and

Whereas, Now such needed requirements are imposed by some major hospitals in Tennessee; and

Whereas, Limits of medical liability insurance do not correlate with a physician's ability, training, character, or ethical behavior, which are the traditional requirements for staff appointments; and

Whereas, The increases in premiums for greater limits of insurance will be passed on to patients at a time when physicians are attempting and being forced to hold costs down. Now, therefore be it

RESOLVED, That the Tennessee Medical Association encourage hospital presidents and executive boards not to require limits of professional liability insurance without approval by the medical staff as a prerequisite for medical staff privileges; and be it further

RESOLVED, That this resolution be sent to all hospital presidents in Tennessee, the boards of directors of hospitals, and appropriate insurance carriers; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE C—*recommended adoption of Resolution No. 16-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 17-87

Peer Review Organization (PRO) Sanctions

By: C. Eugene Jabbour, M.D.
Memphis-Shelby County Medical Society

Whereas, Recent activities of the Peer Review Organizations (PROs) have included a system of punitive sanctions for physicians; and

Whereas, These sanctions have been distributed locally by the Mid-South Foundation for Medical Care, Inc. in a document entitled, "Intervention of Quality Related Occurrences"; and

Whereas, This document appears to recognize as punishable events a vast array of examples ranging from trivial oversights with no harm to the patient, to catastrophic but recognizably unavoidable consequences of the care of gravely ill patients; and

Whereas, These sanctions can result in untoward financial consequences and possible legal liability to the responsible physicians. Now, therefore be it

RESOLVED, That the Tennessee Medical Association oppose those aspects of the Peer Review Organization (PRO) sanctions which attempt to make the attending physician liable for trivial events or unavoidable catastrophe; and be it further

RESOLVED, That the Tennessee Medical Association House of Delegates instruct the Board of Trustees to establish a task force strongly supported by the staff to monitor the operation of this program and lend aid to Tennessee Medical Association members in appealing such sanctions; and be it further

RESOLVED, That a similar resolution be presented to the American Medical Association by the Tennessee delegation and a copy of this resolution be forwarded to the members of the board of the Mid-South Foundation for Medical Care, Inc. and the Tennessee congressional delegation; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*offered Substitute Resolution No. 8-87 to replace original Resolutions No. 8-87 and No. 17-87; recommended adoption of Substitute Resolution No. 8-87.*

ACTION: **NOT ADOPTED (Was replaced with Substitute Resolution No. 8-87, which was adopted as amended.)**

RESOLUTION NO. 18-87

Mandatory A.M. Admissions

By: Daniel J. Scott, Jr., M.D.
Memphis-Shelby County Medical Society

Whereas, As of the first of the year, 1987, insurance companies have been applying more and more pressure to surgeons to admit patients on the same day they are having major surgery, making it extremely difficult to get the laboratory work, x-rays, medical consultations, and in some cases, second opinions done before late in the afternoon; and

Whereas, This causes considerable inconvenience to the patients, particularly those who are coming from many miles out of town, is medically unsafe, and the only savings for insurance companies is an overnight hotel expense; and

Whereas, The surgeon is placed at an increasing liability for risk to the patient that may be apparent on laboratory or x-ray studies that are not available prior to the surgery. Now, therefore be it

RESOLVED, That the Tennessee Medical Association House of Delegates oppose mandatory A.M. admissions of patients undergoing major surgery because of the increased risk to the patient; and be it further

RESOLVED, That the Tennessee Medical Association Board of Trustees establish an ad hoc committee to coordinate with a Tennessee Hospital Association committee to seek redress through the commissioner of Commerce and Insurance in Tennessee; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE C—*recommended adoption of Resolution No. 18-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 19-87

Reversal of Roe vs Wade

By: Robert P. N. Shearin, M.D.
Memphis-Shelby County Medical Society

Whereas, The American Medical Association in 1859 unanimously adopted a report on abortion which termed it "unwarrantable destruction of human life"; and

Whereas, The American Medical Association in 1871 unanimously adopted a report on abortion which termed it "the wholesale destruction of unborn infants"; and

Whereas, The unborn child has, in recent years, been even more clearly recognized, by physicians and lay public alike, as a patient separate from the mother and, as such, subject to independent medical and surgical treatment; and

Whereas, Since the *Roe vs Wade* decision, which legalized elective abortion, more than 19 million unborn children have been killed; and

Whereas, Supreme Court Justice Sandra Day O'Connor has stated that the *Roe vs Wade* decision cannot endure because it is "on a collision course with itself . . ." due, in part, to scientific progress which recognizes and protects the living unborn child even earlier in gestation; and

Whereas, Increasing public awareness that abortion is a killing procedure performed by physicians diminishes the public's respect for Medicine as a healing profession; and

Whereas, The American Medical Association has rightly considered the Hippocratic Oath as a keystone of its opposition to medical participation in legal executions by lethal injections. Now, therefore be it

RESOLVED, That the Tennessee Medical Association hereby call upon the American Medical Association, through its House of Delegates and all other lawful means available, to restore public confidence in Medicine as a healing profession which consistently rejects involvement in killing, by urging all physicians to work for the reversal of the *Roe vs Wade* decision; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE B—*recommended non-adoption of Resolution No. 19-87.*

ACTION: **NOT ADOPTED**

RESOLUTION NO. 20-87

Retrospective Review and Physician Responsibility

By: C. Eugene Jabbour, M.D.
Memphis-Shelby County Medical Society

Whereas, We are now seeing the Peer Review Organization (PRO) regulations on clinical practice in the form of preadmission certification of hospital admissions and retroactive review and denial of hospitalizations; and

Whereas, There has so far been no documented evidence that this system either maintains optimal quality of care or results in cost reduction; and

Whereas, The patient's geographical location in regard to distance of his home from the hospital and his personal circumstances with regard to availability of family for one's home care do not appear to enter the decision of whether inpatient care is allowed; and

Whereas, That patient welfare and quality of care must be of paramount importance both within and outside of the Medicare system, and that the physician must act as the patient's advocate within this system; and

Whereas, The current situation leaves the physician in the untenable position of being subject to censure if he admits a patient too readily and has that admission later denied, or to sanction with economic and punitive consequences if he too rapidly discharged a patient; and

Whereas, The current system of retroactive denial involves communication between the PRO and the patient with no explanation of the specific reasons for denial to either physician or patient; and

Whereas, The present trends in Medicare reimbursement are leading toward a rationing of care to the Medicare recipient and indeed toward a two tiered system of health care. Now, therefore be it

RESOLVED, That organized medicine offer input to the Peer Review Organization (PRO) in attempting to modify the retrospective regulations as they stand now, recognizing that a review organization made up of our peers is far preferable to one made up of non-physicians; and be it further

RESOLVED, That a copy of this resolution be forwarded to the American Medical Association, the Tennessee congressional delegation, the Health Care Financing Administration, and the Mid-South Foundation for Medical Care, Inc.; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*offered Substitute Resolution No. 12-87 to replace original Resolutions No. 12-87 and No. 20-87; recommended adoption of Substitute Resolution No. 12-87.*

ACTION: **NOT ADOPTED (Was replaced with Substitute Resolution No. 12-87, which was adopted.)**

RESOLUTION NO. 21-87

Preferred Provider Organizations (PPOs)

BY: THOMAS W. CURREY, M.D., PRESIDENT
CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY

Whereas, The number of Tennesseans receiving medical and health care services through employer sponsored HMOs, PPOs, and other alternative delivery systems is increasing; and

Whereas, These plans vary significantly regarding organizational, financial, and benefits structures; and

Whereas, There is increasing competition among these various plans in an effort to achieve significant market share; and

Whereas, This competition has resulted in plans having suffered financial losses, reduced or altered benefits, or discontinuation of operation; and

Whereas, Tennesseans receiving health and medical care through employer sponsored plans may have limited choice among plans offered, may have to alter existing physician-patient relationships as a condition for participation in various plans, and may have to change plans due to changed employment status, changed employer plans, or termination of a particular plan; and

Whereas, Preferred Provider Organizations (PPOs) are not specifically regulated in Tennessee. Now, therefore be it

RESOLVED, That the Tennessee Medical Association seek the support of employers, hospitals, and appropriate others in developing of legislation in Tennessee, specifically regulating Preferred Provider Organizations (PPOs); and be it further

RESOLVED, That the Tennessee Medical Association work with the Tennessee Department of Commerce and Insurance in development of such legislation and regulations; and be it further

RESOLVED, That such legislation establish requirements for fair and objective credentialing of participating providers and establish other appropriate measures to assure high quality care and service; and be it further

RESOLVED, That the Tennessee Medical Association cooperate with the Tennessee Department of Commerce and Insurance in monitoring the care and service provided by all benefit plans to Tennesseans; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*recommended referral of Resolution No. 21-87 to the TMA Medical Practice Committee for further study.*

ACTION: **REFERRED TO TMA MEDICAL PRACTICE COMMITTEE**

RESOLUTION NO. 22-87

Health Insurance

BY: JOSEPH L. WILLOUGHBY, M.D.
WILLIAMSON COUNTY MEDICAL SOCIETY

Whereas, Health insurance has made radical changes in the past several years, and continues to change, making it more difficult for the average individual to understand the technical aspects of his policies; and

Whereas, Health insurance is now having a major impact on the quality and accessibility of health care, and with increasing frequency, the health plans are moving toward rationing of care; and

Whereas, Many of these plans interfere with the day-to-day practice of medicine requiring more administrative expenses for the patient and office force, with little productivity toward saving health care dollars. Now, therefore be it

RESOLVED, That the Tennessee Medical Association, through its component societies and individual members, publicly acknowledge support of health insurance plans that provide quality coverage; and be it further

RESOLVED, That Tennessee Medical Association support be acknowledged through the media, personal contact, and taking a position of opposition against plans that manipulate quality health care; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE C—*recommended adoption of Resolution No. 22-87 as amended.*

ACTION: **REFERRED TO TMA BOARD OF TRUSTEES**

RESOLUTION NO. 24-87

M.D. Identification

BY: PARKER D. ELROD, M.D.
BUFFALO RIVER VALLEY MEDICAL SOCIETY

Whereas, Hospitals and medical staff are continually faced with applications from doctors for hospital privileges; and

Whereas, There is no satisfactory way to verify the applicant without pictorial identification; and

Whereas, It is a readily recognized fact that many individuals each year fraudulently pass themselves off as being medical doctors. Now, therefore be it

RESOLVED, That the medical schools be encouraged to develop a passport type identification for students with photo and fingerprints that can be used throughout their careers as identification; and be it further

RESOLVED, That this resolution be presented to the American Medical Association; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE D—*recommended referral of Resolution No. 24-87 to the TMA Board of Trustees for further study.*

ACTION: **REFERRED TO TMA BOARD OF TRUSTEES**

RESOLUTION NO. 25-87

Formation of Medical Student Section

By: Tom Thompson, Student Delegate
Memphis-Shelby County Medical Society

Whereas, Medical students in the state of Tennessee have a genuine interest in the activities of organized medicine within the state and the nation; and

Whereas, The need for unity within the medical profession is ever increasing; and

Whereas, The American Medical Association formed a Medical Student Section in 1972 giving students a voting delegate in the AMA House of Delegates; and

Whereas, The American Medical Association has a policy that active participation by students is vital to the future of the Federation and the unity of the medical profession. Now, therefore be it

RESOLVED, That the Tennessee Medical Association form a Medical Student Section to provide a forum within the Tennessee Medical Association for the exchange of information among students in medical school and their more senior colleagues; and be it further

RESOLVED, That the Tennessee Medical Association include medical students in the decision or policy-making process affecting all physicians in the state of Tennessee; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE D—*offered Substitute Resolution No. 14-87 to replace original Resolutions No. 14-87 and No. 25-87; recommended adoption of Substitute Resolution No. 14-87.*

ACTION: **NOT ADOPTED (Was replaced with Substitute Resolution No. 14-87, which was adopted.)**

RESOLUTION NO. 26-87

Reentry Assistance Needed by Impaired Physicians

By: Arden J. Butler, Jr., M.D., Chairman
TMA Board of Trustees

Whereas, Appropriate treatment for certain physician impairment requires a minimum of four months out of active medical practice; and

Whereas, By necessity, total dedication to treatment issues during this time is required of each physician; and

Whereas, Such total dedication leaves no time for continuing medical education activities or technical practice, such as scrubbing for surgery or assisting in surgery for the duration of the treatment process; and

Whereas, Such deficits created by the treatment process have a psychological and practical negative impact upon both physician and his reentry into the medical community. Now, therefore be it

RESOLVED, That the Tennessee Medical Association endorse efforts by the Impaired Physician Peer Review Committee to develop a reentry professional support network that would effectively deal with practice deficits, including assisting in surgery, participating in medical staff seminars, and other continuing medical education activities in areas not necessarily the home community; and be it further

RESOLVED, That this support network include utilizing certain medical specialists and willing members of the Tennessee Medical Association to assist in implementation of the reentry support network; and be it further

RESOLVED, That such a plan, when finally formulated, be approved by the Board of Trustees of the Tennessee Medical Association prior to implementation; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE D—*recommended adoption of Resolution No. 26-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

RESOLUTION NO. 27-87

Indigent Care—Medical Home—Volunteerism

By: Arden J. Butler, Jr., M.D., Chairman
TMA Board of Trustees

Whereas, The Tennessee Medical Association and its member physicians are committed to the proposition that every Tennessean should have good quality medical care; and

Whereas, Inpatient, indigent medical care is presently being addressed by many public forums, but ambulatory indigent care with its cost saving potential is not; and

Whereas, A "medical home" model has been established by the Tennessee Chapter of the American Academy of Pediatrics (TCAAP) and the Department of Health and Environment (DHE) in which rosters of physicians who will see indigent children are kept by local county health departments; and

Whereas, Presently, 714 pediatricians and family physicians across the state are seeing these children who have no physician and accepting them whether they are private third-party pay or indigent. Now, therefore be it

RESOLVED, That the Tennessee Medical Association strongly urge its component medical societies to set up "medical homes" rosters for indigent adults, similar to those presently in place for children; and be it further

RESOLVED, That the presidents of the component societies work with the commissioner of the Department of Health and Environment, the directors of the county and regional health departments, and the Primary Care Liaison Committee of the Tennessee Medical Association in setting up these medical homes; and be it further

RESOLVED, That all physicians be encouraged to communicate and work with the public sector in a spirit of volunteerism to alleviate the problems of indigent care in Tennessee; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*recommended adoption of Resolution No. 27-87 as amended.*

ACTION: **ADOPTED AS AMENDED**

SUBSTITUTE RESOLUTION NO. 28-87

Public Information on Health Care

By: Thurman L. Pedigo, M.D.
Rural Caucus

Whereas, There is increasing interference in the physician-patient relationship by third parties, including government, health care administrators, insurance companies and other regulatory bodies; and

Whereas, This interference impedes delivery of quality patient care in Tennessee; and

Whereas, Existing legislation mandates this interference; and

Whereas, An informed public can greatly influence legislative action and conduct of health care providers and regulators; and

Whereas, Physicians lack an adequate media forum for expression of their viewpoint. Now, therefore be it

RESOLVED, That the Tennessee Medical Association Board of Trustees appoint a task force to develop a plan to create a public information campaign that would outline interference in the physician-patient relationship by government and third party providers, and seek to develop public support for elimination of that interference; and be it further

RESOLVED, That the task force report its findings and recommendations within six months to the Board of Trustees of the Tennessee Medical Association; and be it further

RESOLVED, That available resources be used to implement the recommendations of the task force; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE D—*offered Substitute Resolution No. 28-87 in place of original Resolution No. 28-87; recommended adoption of Substitute Resolution No. 28-87.*

ACTION: **ADOPTED**

RESOLUTION NO. 29-87

Additional Trauma Center Designation

By: John Standridge, M.D.

Whereas, The state of Tennessee recognizes the need for efficient and effective care of the multiply traumatized patient; and

Whereas, Tennessee is noted to be a state with much of the population located outside of urban centers; and

Whereas, It has been noted by some authors that trauma care in non-urban settings is best accomplished by efficient stabilization in local emergency rooms and transfer of patients, if necessary, to appropriate facilities; and

Whereas, Tennessee has multiple facilities effective in the treatment of trauma, some of which will not be designated as trauma centers under the current Licensing Board for Health Care Facilities proposal. Now, therefore be it

RESOLVED, That the Licensing Board for Health Care Facilities designate a third level of trauma center inclusive in Level I, Level II and Level III, recognizing expertise in trauma care of facilities where it exists; and be it further

RESOLVED, That this resolution, unless reaffirmed or modified prior thereto, shall terminate after the regular annual meeting of the House of Delegates in 1994.

REFERENCE COMMITTEE A—*recommended referral of Resolution No. 29-87 to the TMA Board of Trustees.*

ACTION: **REFERRED TO TMA BOARD OF TRUSTEES**

COMMUNITY SERVICE AWARDS

Three outstanding Tennessee citizens with impressive records of public service have been presented TMA's 1987 Community Service Awards. The awards were presented on April 8, 1987, by TMA Board of Trustees Chairman Arden J. Butler, Jr., M.D.

Jerry LeBron Bell is an associate professor of health and physical education at Tennessee Technological University in Cookeville. He has been personally involved with the Special Olympics Program as area coordinator for the past 13 years. Under his direction, Cookeville has hosted the Special Olympic bowling tournament for the past nine years and the soccer tournament for the past two years. He has also been involved in Special Olympics volleyball, basketball, and swimming.

Mr. Bell took participants to the International Special Olympic Games in Rockport, New York, in 1979 and to Baton Rouge, Louisiana, in 1983. This year, he will take a group to the Special Olympic Games in South Bend, Indiana. He is an active fundraiser promoting the Special Olympics to civic clubs, businesses, industries, and other organizations of the Upper Cumberland area.

Further, Mr. Bell promotes track meets for youth, having organized and coordinated the spring track meet for elementary schools throughout Putnam County with the cooperation of the local school system and Tennessee Tech. This program encourages participation and interest in track by elementary school aged children.

Barbara Ross Moore of Jackson is the founder of the Jackson Host Home Program, in cooperation with Jackson-Madison County General and HCA Regional hospitals. She currently serves as volunteer coordinator of the program.

Mrs. Moore became familiar with the host home concept in 1984 when her son was critically injured in an automobile accident in Savannah, Georgia. She and her husband were recipients of a host home for five weeks, and learned the necessity of family care in the team effort of treating a patient both physically and emotionally. She brought the concept to Jackson upon her return.

The Jackson Host Home Program provides housing and supportive companionship in a home-like environment for out-of-town relatives of seriously ill or injured patients. The emotional and spiritual support from the program helps ease the burden and stress a family experiences while a loved one is hospitalized.

Mrs. Moore arranges lodging for the family, visits the family daily offering assistance, and ensures that necessary tasks, such as laundry, are done. She has recruited 47 host homes, and has enlisted the participation of nine churches, seven restaurants, and three florist/nurseries. She expects this program to continue because of the generosity and love the people of Jackson extend to the family members. Her goal is to start host home programs in other major Tennessee cities.

Mrs. Moore has received the "Good News Award of the Week" from WLJT-TV at the University of Tennessee in Martin. She currently serves on the Jackson Volunteer Clearinghouse Board of Directors.

Suzanne Sebert of Knoxville is the Director of Community Services for the Knox County Department of Welfare and Institutions. She is credited with maintaining the availability and quality of medical care when the Knox County Indigent Care Program had extensive budget cuts. Her efforts and talent allowed the program to continue successfully, and the program has received national recognition.

Mrs. Sebert is the chairperson for the Social Services Regional Advisory Council, and serves on the Social Services State Advisory Council, the Knox County Indigent Care Advisory Committee, and is a board member of the Community Action Committee.

She is an advisory member of the Emergency Medical Services Committee of the Knoxville Academy of Medicine. She was the government division head for the United Way Campaign in 1986 and a member of the United Way Long-Range Strategy Committee. She has been a Girl Scout leader since 1983, is involved with the AFS International Scholarships, and is currently a participant in the Leadership Knoxville Class of 1987.

TENNESSEE'S OUTSTANDING PHYSICIAN OF THE YEAR

Thomas K. Ballard, M.D.

The TMA House of Delegates selected Thomas K. Ballard, M.D. of Jackson as the 1987 Outstanding Physician of the Year at the 152nd TMA annual meeting on April 8, 1987. The speaker of the House of Delegates, Malcolm R. Lewis, M.D., presented the award to Dr. Ballard on April 11 during the closing session of the House of Delegates. Dr. Ballard was nominated for the award by the Consolidated Medical Assembly of West Tennessee.

Thomas K. Ballard, M.D., a native of St. Joe, Texas, received his medical degree from the University of Tennessee College of Medicine in 1949. After completing a one-year internship at Baptist Memorial Hospital in Memphis, he returned to Jackson to practice medicine, serving as city physician of Jackson until 1958.

In nominating Dr. Ballard for this award, the Consolidated Medical Assembly of West Tennessee said "Thomas K. Ballard, M.D. represents the epitome of a caring physician who has been not only active throughout his entire professional career in the practice of medicine, but also has been exceedingly active in the affairs of organized medicine at all levels . . ."

Dr. Ballard was president of the TMA in 1984-85, and has been a member of the TMA House of Delegates since 1966. He has served many years as a member of the TMA Board of Trustees, and was a member of the Independent Medicine's Political Action Committee—Tennessee (IMPACT) Board of Directors from 1979-85. He has been a member of the TMA Interprofessional Liaison Committee since 1964, serving as its chairman since 1975, and he also currently serves on the TMA Peer Review and Continuing Medical Education committees. He is a delegate to the AMA House of Delegates and a member of the AMA Advisory Committee for Continuing Medical Education.

He was president of the Consolidated Medical Assembly of West Tennessee in 1970, and has been a member of its House of Delegates since 1960. He was a member of its Judicial Council from 1971-73 and served as its chairman in 1973.

Since 1960, Dr. Ballard has been a member of the Board of Directors and the Congress of Delegates of the Tennessee Academy of Family Physicians. He is a former president of the Academy, and was elected Tennessee's Family Physician of the Year in 1975. He is a Charter Fellow of the American Academy of Family Physicians and is a member of the Academy's House of Delegates.

Outstanding Physician of the Year Award is presented to Dr. Thomas K. Ballard, Jackson (right) by TMA House of Delegates speaker, Dr. Malcolm R. Lewis.

Dr. Ballard was a founding member of the 17-county West Tennessee Health Improvement Association, and he served on its Board of Directors and Executive Committee from 1974-82, acting as treasurer from 1974-79. He was a member of the Board of Directors of the Mid-South Foundation for Medical Care from 1977-85, and has been a member of its Quality Review Committee since 1978.

Dr. Ballard served as chairman of the State of Tennessee Public Health Council from 1972-75, as a member of the Hospital Licensing Board from 1972-75, and has been a member of the Medical Advisory Board to the Tennessee Highway Patrol since 1972. He was also a member of the Governor's Select Committee on Health Care Cost Containment in 1984-85.

He has served Madison County as a member of its Board of Health since 1965 and has been its chairman since 1967. He has been county medical examiner since 1962, and county coroner since 1972.

His civic endeavors have been equally extensive with prominent service to a number of organizations. He has been an active leader of the Lambuth Memorial United Methodist Church of Jackson, serving as chairman of the Administrative Board for 12 years, past chairman of the Board of Trustees, and a member of the Pastor-Parish Relations Committee.

He is married to the former Mary Frances Bond of Jackson, and they have three children and five grandchildren.

DISTINGUISHED SERVICE AWARDS

Distinguished Service Award recipients (left to right) Drs. James W. Hays, Nashville; Howard R. Foreman, Nashville; and David H. Turner, Chattanooga, with chairman of the TMA Board of Trustees, Dr. Arden J. Butler, Jr.

The Distinguished Service Award, established in 1964, is presented annually by the TMA Board of Trustees to physician members in recognition of outstanding service or contributions to the advancement of medical science, the TMA, or the public welfare whether of a civic or scientific nature. At the TMA's 152nd annual meeting in Knoxville, Arden J. Butler, Jr., M.D., chairman of the Board, presented three recipients with this award for 1987.

Howard Ray Foreman, M.D., a practicing internist in Nashville, has served as a member of the Tennessee Board of Medical Examiners for the past 15 years. He has served as Secretary to the Board for the past 12 years.

"I know of no other contribution made by a physician of this state which requires more effort, time, judgment, and self-sacrifice than that of being a member of the Board of Medical Examiners, and especially being the (Board's) Secretary," stated John H. Burkhart, M.D. of Knoxville in supporting Dr. Foreman's nomination for this award.

Born in Mitchell, South Dakota, in 1920, Dr. Foreman received his medical degree from the Vanderbilt University School of Medicine in 1950. He was chief medical resident at St. Thomas Hospital in 1973.

Dr. Foreman is associate clinical professor of medicine at Vanderbilt University School of Medicine, and is affiliated with Baptist, West Side, Park View, and St. Thomas Hospitals in Nashville.

James W. Hays, M.D., a practicing neurosurgeon in Nashville, was nominated for this award by the Nashville Academy of Medicine. He has served as chairman of TMA's Committee on Legislation for ten years, and has always brought a level of importance and enthusiasm to his role as "chief physician monitor" of the state legislature.

Dr. Hays was president of TMA in 1979-80. He has served as president of the Baptist Hospital (Nashville) medical staff, a member of the Board of State Volunteer Mutual Insurance Company, chairman of the Board of TMA, and president of the Nashville Academy of Medicine. He has also served as a member of the Tennessee Primary Care Advisory Commission.

Born in South Williamson, Kentucky, in 1931, Dr. Hays received his medical degree from Vanderbilt University School of Medicine in 1957. He completed his internship and general surgery and neurosurgery residencies at Vanderbilt University Hospital.

Dr. Hays is a clinical instructor of neurological surgery at Vanderbilt University School of Medicine, and is affiliated with Baptist, St. Thomas, Vanderbilt University, Park View, and West Side Hospitals in Nashville.

David H. Turner, M.D., a practicing ophthalmologist in Chattanooga, was nominated for this award by the Chattanooga-Hamilton County Medical Society. He has served as chairman of the Tennessee Public Health Council and as a member of the Tennessee Licensing Board for Health Care Facilities since 1979.

Dr. Turner served on numerous committees in the TMA and in his component society. He was president of the TMA in 1977-78, and retired as a delegate to the American Medical Association House of Delegates this past year, having served since 1971. He has served as secretary-treasurer and president of the Tennessee Academy of Ophthalmology.

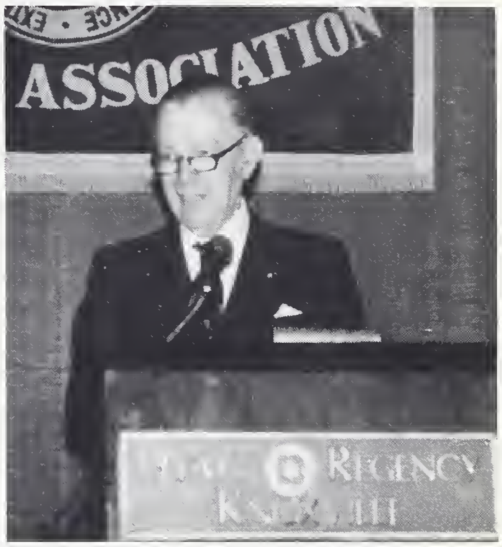
Born in Jasper, Tennessee, in 1927, Dr. Turner received his medical degree from the University of Tennessee College of Medicine in 1952, and completed his internship the following year at Baroness Erlanger Hospital in Chattanooga. From 1954-57, he operated a general practice in Jasper and then went on to complete his residency in ophthalmology at Emory University in Atlanta.

Dr. Turner is a Diplomate of the American Board of Ophthalmology and a Fellow of the American Academy of Ophthalmology and the American College of Surgeons.

TMA Annual Meeting Highlights
Knoxville—April 1987

IMPACT Luncheon speaker Mr. Michael E. Dunn addressed the role of the physician involvement in the political process.

Outgoing TMA president Dr. James R. Royal, Chattanooga (left) relinquishing gavel to incoming president Dr. James T. Galyon, Memphis.



Medicine and Religion Breakfast speaker Dr. Paul Wilson Brand spoke on "Men and Women in the Image of God."

TMA president Dr. James R. Royal gives a "thumbs up" while being serenaded by members of the Chattanooga Singers from UT Chattanooga.

Dr. and Mrs. James T. Galyon and Dr. and Mrs. James R. Royal at the annual President's Banquet.

Enjoying a light moment before the second session of the House of Delegates convenes are (left to right) Mr. L. Hadley Williams, TMA executive director; Dr. Malcolm R. Lewis, Nashville, outgoing speaker of the House of Delegates; and Dr. F. Hammond Cole, Jr., newly elected speaker of the House.

1987 TMA Annual Meeting—House of Delegates Composition

First Session: April 8—Second Session: April 11

EX-OFFICIO MEMBERS

OFFICERS

		First Session	Second Session
President	Jomes R. Royol, M.D.	Present	Present
President-Elect	Jomes T. Golyon, M.D.	Present	Present
Vice-President	Jocob T. Brodsher, Jr., M.D.	Present	Present
Vice-President	Horry E. Burck, Jr., M.D.	Present	Present
Vice-President	Kenneth R. Moloney, M.D.	Present	Present

BOARD OF TRUSTEES

	First Session	Second Session
John S. Buchignoni, M.D.	. . . .	. . . .
Arden J. Butler, Jr., M.D.	Present	Present
Jock Butterworth, Jr., M.D.	Present	. . . .
Dee J. Conale, M.D.	Present	Present
F. Hammond Cole, Jr., M.D.	Present	Present
David G. Gerkin, M.D.	. . . .	. . . .
Robert W. Ikard, M.D.	. . . .	Present
Chorles E. Jordon, III, M.D.	Present	Present
Molcolm R. Lewis, M.D.	Present	Present
Hoys Mitchell, M.D.	Present	. . . .
Clarence R. Sonders, M.D.	Present	Present
Paul R. Stumb, M.D.	Present	. . . .

COUNCILORS

		First Session	Second Session
1st District	Chorles E. Allen, M.D.	Present	Present
2nd District	John E. Kesterson, M.D.	Present	Present
3rd District	Robert W. Myers, Sr., M.D.	. . . .	. . . .
4th District	Will G. Quorles, Jr., M.D.	. . . .	. . . .
5th District	Sue P. W. Johnson, M.D.	. . . .	. . . .
6th District	Howord L. Solyer, M.D.	Present	Present
7th District	Tom R. Duncon, M.D.	Present	Present
8th District	Montie E. Smith, Jr., M.D.	Present	Present
9th District	Jomes H. Rogsdale, M.D.	Present	Present
10th District	Dennis A. Higdon, M.D.	Present	Present

AMA DELEGATES

	First Session	Second Session
Thomos K. Bollord	Present	Present
John S. Derryberry, M.D.	Present	Present
Hamel B. Eoson, M.D.	Present	Present
William O. Miller, M.D.	Present	. . . .
John B. Thomison, M.D.	Present	Present
A. Roy Tyrer, Jr., M.D.	Present	Present

MEMBER OF AMA JUDICIAL COUNCIL

	First Session	Second Session
John H. Burkhart, M.D.	Present	Present

PAST PRESIDENT OF AMA

	First Session	Second Session
Tom E. Nesbitt, M.D.	. . . .	Present

PAST PRESIDENTS OF TMA

	First Session	Second Session
E. Kent Corter, M.D.	Present	. . . .
J. Kelley Avery, M.D.	Present	Present
David H. Turner, M.D.	Present	Present
Jomes W. Hoys, M.D.	Present	Present
George A. Zirkle, Jr., M.D.	Present	Present
Allen S. Edmonson, M.D.	Present	Present
Not E. Hyder, Jr., M.D.	Present	. . . .

CHIEF MEDICAL OFFICER DEPARTMENT OF HEALTH AND ENVIRONMENT

	First Session	Second Session
Fredio S. Wodley, M.D.	Present	Present

DELEGATES

EAST TENNESSEE GRAND DIVISION

County Society		First Session	Second Session
BLOUNT	Marvin D. Peterson, M.D.	Present	Present
	John J. Ingrom, III, M.D.	Present	Present
BRADLEY	John Stondridge, M.D.	Present	Present
	John W. Chombers, M.D.	Present	Present
CAMPBELL	Elijoh G. Cline, Jr., M.D.	Present	Present
CARTER	Gory R. Chombers, M.D.	Present	Present
CHATTANOOGA-HAMILTON	Thomos W. Currey, M.D.	Present	Present
	Robert K. Berglund, M.D.	Present	Present
	Dovid R. Bornes, M.D.	Present	Present
	Rolph E. Nipp, M.D.	Present	Present
	Pete S. Soteres, M.D.	Present	Present
	R. Phillip Burns, M.D.	Present	Present
	Joel E. Avery, M.D.	Present	Present
	Somuel L. Bonks, M.D.	. . . .	Present
	C. Windom Kimsey, M.D.	. . . .	Present
	Alfred Rogers, M.D.	. . . .	Present
	Williom E. Rowe, M.D.	. . . .	Present
	Myron J. Szczukowski, Sr., M.D.	. . . .	Present
COCKE*	Steven Gore, M.D.	. . . .	. . . .
CUMBERLAND	Donathon M. Ivey, M.D.	Present	Present
GREENE	William J. Smeod, M.D.	. . . .	. . . .
	Larry E. Rodgers, M.D.	. . . .	. . . .
HAWKINS*			
KNOXVILLE ACADEMY	R. Kent Farris, M.D.	Present	Present
	Ted F. Hoase, Jr., M.D.	Present	Present
	Joseph B. Moon, M.D.	Present	Present
	Trovis E. Morgon, M.D.	Present	Present
	George H. Wood, M.D.	Present	Present
	Williom D. Block, M.D.	Present	Present
	A. B. Kliefoth, III, M.D.	Present	Present
	Carroll W. McGinnis, M.D.	Present	Present
	Randolph M. Lowry, M.D.	Present	. . . .
	R. Leslie Horgrove, M.D.	Present	. . . .
	Mory B. Duffy, M.D.	Present	. . . .
	Michoel M. Miller, M.D.	. . . .	Present
	Williom G. Loing, M.D.	. . . .	Present
LAKEWAY	William J. Gutch, III, M.D.	Present	Present
	Jose G. Amodor, Jr., M.D.	. . . .	. . . .
McMINN	Clyde Mortin, M.D.	. . . .	Present
MONROE	Fronk H. Lowry, M.D.	. . . .	Present
ROANE-ANDERSON	William E. Bennett, M.D.	Present	Present
	Robert R. Cosey, M.D.	Present	Present
	Chorles B. Gurney, M.D.	. . . .	Present
SCOTT	Timothy F. Gorner, M.D.	Present	Present
SEVIER	Vincent B. Tolley, M.D.	Present	Present
SULLIVAN	Hal S. Stubbs, M.D.	Present	Present
	J. Lowrence Joyne, Jr., M.D.	Present	Present
	Billy N. Golden, M.D.	Present	Present
	Richord D. Boker, M.D.	Present	Present
	Donold W. Boles, M.D.	Present	Present
	Jere W. Ferguson, M.D.	Present	. . . .
	Lorenzo D. Stroder, M.D.	. . . .	Present
WASHINGTON-UNICOI-JOHNSON	Duane C. Budd, M.D.	Present	Present
	Clorence E. Goulding, Jr., M.D.	Present	Present
	W. Richord McGowan, Jr., M.D.	Present	Present
	Burgin E. Dossett, Jr., M.D.	. . . .	Present
	Loren J. Mouw (student delegate)	. . . .	Present

MIDDLE TENNESSEE GRAND DIVISION

County Society		First Session	Second Session
BEDFORD	John S. Derryberry, M.D.	Present	Present
BENTON-HUMPHREYS	Subhi D. S. Ali, M.D.	. . . .	Present
BUFFALO RIVER VALLEY	Porker D. Elrod, M.D.	Present	Present
COFFEE	Chorles H. Webb, M.D.	Present	Present
DeKALB	Melvin L. Blevins, M.D.	. . . .	Present
DICKSON	Venk Moni, M.D.	. . . .	. . . .
FENTRESS*			
FRANKLIN	M. Dovid Stockton, M.D.	. . . .	. . . .
GILES*	J. Vonce Fentress, M.D.	. . . .	. . . .
JACKSON*			
LAWRENCE	J. Carmock Hudgins, M.D.	. . . .	Present
LINCOLN	Joseph F. Rolston, Jr., M.D.	Present	Present
MACON*			
MARSHALL	Kenneth J. Phelps, Jr., M.D.	Present	. . . .
MAURY	John O. Willioms, Jr., M.D.	Present	Present
	Lowrence R. Nickell, M.D.	Present	Present
MONTGOMERY	Williom J. Pedigo, M.D.	. . . .	Present
	Tony J. Montgomery, M.D.	Present	Present
NASHVILLE ACADEMY	Louis J. Bernard, M.D.	Present	Present
	H. Victor Broren, M.D.	Present	Present
	Roy G. Hommonds, M.D.	Present	Present
	Dovid E. McKee, M.D.	Present	Present
	Cullen R. Merritt, II, M.D.	Present	Present
	Ronold Overfield, M.D.	Present	Present
	R. Benton Adkins, M.D.	Present	Present
	John Lomb, M.D.	Present	Present
	Henry Foster, M.D.	Present	Present
	Richord Light, M.D.	Present	Present
	Howord S. Kirshner, M.D.	Present	. . . .
	Soroh H. Sell, M.D.	Present	. . . .
	C. Gordon Sell, M.D.	Present	. . . .
	Frank Perry, M.D.	Present	. . . .
	Somuel H. Dillord, Jr., M.D.	. . . .	Present
	John L. Forringer, Jr., M.D.	. . . .	Present
	Williom B. Horwell, Jr., M.D.	. . . .	Present
	Jomes M. High, M.D.	. . . .	Present
	Kent Kyger, M.D.	. . . .	Present
	Ann H. Price, M.D.	. . . .	Present
	Joseph W. Scobey, M.D.	. . . .	Present
	Michoel B. Seshul, M.D.	. . . .	Present
	J. C. Pennington, Jr., M.D.	. . . .	Present
	Joe M. Chisholm, Jr., M.D.	. . . .	Present
	Koren B. Duffy, M.D.	. . . .	Present
	Michoel Volley (student delegate)	Present	Present
OVERTON	Michoel Cox, M.D.	. . . .	. . . .
PUTNAM	Randoll G. Somples, M.D.	Present	Present
ROBERTSON	John B. Turner, M.D.	. . . .	. . . .
RUTHERFORD/STONES RIVER ACADEMY	Rodney Bryont, M.D.	Present	Present
	Olin Willioms, M.D.	Present	Present
SMITH	Hugh E. Green, M.D.	Present	Present
SUMNER	Lloyd T. Brown, M.D.	Present	Present
WARREN	Thurmon L. Pedigo, M.D.	Present	Present
WHITE*			
WILLIAMSON	Joseph L. Willoughby, M.D.	Present	Present
WILSON	Jomes C. Brodshow, Jr., M.D.	Present	. . . .

WEST TENNESSEE GRAND DIVISION

County Society		First Session	Second Session
CONSOLIDATED	Jomes T. Croig, Jr., M.D.	Present	Present
	Oscor M. McCallum, M.D.	Present	Present
	Charles W. White, M.D.	Present	Present
	Jomes H. Donnell, M.D.	Present	. . . .
	Michoel A. McAdoo, M.D.	Present	Present
	Lee Rush, Jr., M.D.	. . . .	Present
HARDIN	H. Blonkenship, M.D.	. . . .	. . . .
HENRY*			
MEMPHIS-SHELBY	Rex A. Amonette, M.D.	Present	Present
	Thomos A. Currey, M.D.	Present	Present
	McCarthy DeMere, M.D.	Present	Present
	Leonord H. Hines, M.D.	Present	Present
	C. Eugene Jobbour, M.D.	Present	Present
	Evelyn B. Ogle, M.D.	Present	Present
	Phil E. Orpet, Jr., M.D.	Present	Present
	Phillip A. Pedigo, M.D.	Present	Present
	William T. Satterfield, Jr., M.D.	Present	Present
	Doniel J. Scott, Jr., M.D.	Present	Present
	Jesse C. Woodoll, Jr., M.D.	Present	Present
	Robert C. McEwon, Jr., M.D.	Present	Present
	Robert P. N. Sheorin, M.D.	Present	Present
	Potrick J. Murphy, M.D.	Present	Present
	James C. Fleming, M.D.	Present	Present
	Shermon Kahn, M.D.	Present	Present
	Jomes W. Pate, M.D.	. . . .	Present
	Richord M. Peorson, M.D.	. . . .	Present
	Albert J. Grobmyer, III, M.D.	. . . .	Present
	Gole Gordner, M.D.	. . . .	Present
	Lee R. Morisy, M.D.	. . . .	Present
	Tom Thompson (student delegate)	Present	Present
NORTHWEST	James W. Shore, M.D.	Present	Present
	Hobort H. Beole, M.D.	Present	Present
TIPTON	Worren A. Alexonder, M.D.	. . . .	. . . .
HOSPITAL MEDICAL STAFF SECTION	Donold L. Goines, M.D.	Present	Present

Ex-officio delegates serving in more than one capacity are listed only once. The above information was taken from attendance records signed by the delegates.

*The following component society delegates were not eligible for seating due to failure to file the 1986 annual report as required by the TMA Constitution and Bylaws: Cocke, Hawkins, Fentress, Giles, Jackson, Macon, White, Henry.

Prevent recurrences month after month*

ZOVIRAX®
(acyclovir)
CAPSULES

Brief Summary

INDICATIONS AND USAGE: Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psychosocial difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

First Episodes (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

Recurrent Episodes:

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

CONTRAINDICATIONS: Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

WARNINGS: Zovirax Capsules are intended for oral ingestion only.

PRECAUTIONS: General: Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

Drug Interactions: Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the F_1 generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

Pregnancy: ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS — Short-Term Administration: The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

Long-Term Administration: The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

Chronic suppressive therapy for recurrent disease: One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

Intermittent Therapy: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

Patients With Acute or Chronic Renal Impairment: One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance ≤10 ml/min/1.73/m².

HOW SUPPLIED: Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200"- Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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*In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

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REPORTS OF OFFICERS

Report of the President

James R. Royal, M.D.

The year 1986 was a year of change for medicine. We have been forced to reevaluate our traditional ways of practicing medicine. We have accepted the fact that the winds of change will affect our future course. How we adjust to influence those changes will decide our future.

The AMA had as the theme for this year's Leadership Conference "Controlling the Changes." Discussions were held on how we as a profession can best influence the direction of the coming changes for the good of our patients and our profession. We must actively participate.

The *Health Policy Agenda for the American People* has just been completed and published. This forward looking innovative project was initiated by the AMA five years ago. One hundred and seventy-two organizations which included insurers, consumer and business groups, government agencies, the AMA, and other health groups were involved in developing a comprehensive framework for health care policy in the United States. All interests of the American people were represented. The coalition's goal is that "through a combination of federal, state and private sector actions, no American will be denied access to quality medical care because of his or her ability to pay." Although we will not agree with some of the recommendations, we should help implement those parts which best serve to provide the best health care possible for our patients.

A dedicated group of our members has worked long and hard to study, organize and launch the TMA-sponsored Independent Practice Association (IPA) as directed by this House of Delegates. More than 1,100 of our members have joined to participate in this venture. The governing board has been chosen and the company has been incorporated. Feasibility studies have been done by professionals with the conclusion that a viable successful project is possible. We will have a physician oriented and directed option to the myriad of other programs being offered by outsiders.

The Tennessee Association for Civil Justice Reform is functioning well. We have joined with organizations representing all areas of society to work for tort reform in Tennessee. The TMA is not alone this year in working for legislative relief of the mess that is affecting all aspects of our professional and business lives. I hope the coalition will remain viable and active until relief is obtained.

I was privileged to attend both sessions of the AMA House of Delegates as an alternate delegate. I was impressed with the intensity and democratic manner in which debate was conducted in reference committees and on the House floor. I am now more comfortable with our national leadership. Our future lies with our continued participation and support of the AMA.

We still have major problems to be solved, and as the leaders in health care we must guide and direct the changes coming. Indigent care, Medicaid, Medicare, catastrophic and long term care must still be addressed and solutions found. We must participate in controlling the changes. As society comes to grips with the moral and ethical questions posed by rapid developments in health care technology, who should have access, under what circumstances should it be applied or withdrawn, and what are the roles of providers and patients in reaching these decisions?

Research funding is essential to the continued improvement of the health of the American people. We must also address health in the workplace, health education and safety, use of health care benefit packages, and education of health professionals. We shall be busy.

Our TMA staff under the leadership of Mr. Hadley Williams has been super in helping me and the other officers through this unique year of change. We can all be proud of our small, but dedicated and capable staff. Thanks to all of you.

As Dr. Galyon takes the office of president, I wish him well and pledge my wholehearted support as we try to control the changes being forced upon us.

REFERENCE COMMITTEE C—*reviewed the report of the president, expressed its deepest thanks to Dr. Royal for his leadership and work this past year, and recommended that the report be filed.*

Report of the Board of Trustees

Arden J. Butler, Jr., M.D., *Chairman*

The Board of Trustees meets quarterly to conduct the business of the Tennessee Medical Association. Some of its duties are to implement policies established by the House of Delegates, administer its affairs, make appointments and awards, and to consider how this organization can better meet the needs of its members. There are five scheduled meetings of the Board of Trustees each year, two being during the annual meeting in April—first that of the old Board meeting on the day that the meeting starts and the newly elected Board meeting on Saturday after the House of Delegates meeting. Between meetings of the full Board, the Executive Committee, composed of five of the officers of the Association, handles various items of importance referred to the Association.

I shall touch briefly on several of the more important items that were handled this year and then will append a much more detailed list of actions taken by the Board. This list is not designed to be read verbatim but should be scanned when you have a few minutes with the thought in mind of the many complex and varied ways that this Association serves its members.

The Board appointed an implementation committee to study the feasibility of forming a statewide independent practice association (IPA). Dr. Thurman Pedigo of McMinnville was appointed chairman of this committee. This group has worked very diligently throughout the year. An initial signup was started in the summer with a participation fee being collected, and this resulted in over 1,100 people being signed up prior to the first of the year. A contract was entered into with Bay State HMO Management of Cambridge, Mass., and they conducted a very thorough and very comprehensive feasibility and pre-development report which was presented in its final form Feb. 3, 1987. With permission of the Board of Trustees of TMA, this group incorporated as the Tennessee Physicians Practice Association, Inc. in December 1986. This was a non-profit corporation under Tennessee law. The federal status of this has not been determined as yet. This is a very important and very complex activity. The first membership meeting of the IPA will be held on April 11. I would urge all of you who are members to attend if you can.

Another important activity this year has been TMA's participation in the Tennessee Association for Civil Justice Reform, Inc., which is pursuing the very worthwhile task of obtaining tort reform in Tennessee. We have been active as an organization in this group. Many of our members individually have been active, and over 400 of them have signed up as individual members of this association. TMA and TMA members have been directly responsible for raising $44,245 to help in this effort, and it is, and will be, an ongoing activity.

We have also implemented a Hospital Medical Staff Section that has met and elected officers. Their full meeting was March 1, 1987. We look forward to this group being an important source of information and activity for our membership.

TMA Physician Services, Inc., our for-profit corporation that handles insurance programs and other activities, generated over $50,000 in profit this year for the Tennessee Medical Association. This is a great help in the ongoing expenses of the organization and will allow us to provide more service and hold dues increases to an absolute minimum.

The Board has taken actions that have strengthened the Impaired Physician Committee. This committee is doing a tremendous job and is gaining national recognition for its work with impaired physicians. Dr. David Dodd is the full-time medical director. Again this year we won the Tharp Award and have designated that money for the ongoing collection of data and supplying of this data in a statistical form only to the national agency to help improve knowledge about this very crucial and worthwhile activity. The Board also approved a methodology whereby Dr. Dodd can be covered adequately during periods of vacation and can have a reasonable time away from work.

Working with government agencies has been of concern to us this year. The Board of Medical Examiners has put out regulations concerning the taking of x-rays in physician offices. These are ongoing discussions, and the final regulations have not been approved as yet. As chairman of your Board of Trustees, I testified at a public hearing on this subject on March 21, 1987, and I would urge the members to keep up with things in this area. We have also looked into some complaints concerning activities of the Mid-South Foundation for Medical Care, Inc., which is the agency that implements peer review for the Medicare program in the state. There is at least one resolution from the Board and other resolutions from other sources to be presented to this body today on those subjects. This, too, is an ongoing type activity that will require vigilance on our part.

Although 166 separate items of business were brought before the Board during regular quarterly meetings, the following are abstracted highlights of some of the more important items of business acted upon by the Board during the past 12 months:

Second Quarter Meeting—April 9 and 12, 1986

The Board:

- —Approved a request by the TMA Auxiliary for an appropriation of $4,000 for a TMAA-sponsored Teen Health Workshop planned for the fall in Memphis.
- —Presented to Mr. Bill Ellis of Smith, Reed, Thompson & Ellis a resolution of commendation in appreciation for his 21 years of service to TMA as administrator of TMA-sponsored insurance plans.
- —Received a report from Dr. Malcolm R. Lewis on a planned premium increase of 12.3% by SVMIC in 1986, the smallest increase in the company's history. Dr. Lewis noted the resignation of Dr. J. Kelley Avery from the SVMIC staff. A proposal has been submitted to the Tennessee Dental Association for professional liability coverage for dentists.
- —Received a report from TMA Staff Attorney Jack Fosbinder that IMPACT has received AMPAC's first place Sustaining Membership Award.
- —Voted to send a letter to the Health Care Financing Administration in support of the Mid-South Foundation for Medical Care as the PRO for Tennessee.
- —Voted to adopt positions on each resolution submitted to the TMA House of Delegates.
- —Received and approved the first quarter financial statement.
- —Voted to send a letter of endorsement for Dr. John Chapman's candidacy for a position on the AMA Council on Medical Education.
- —Received two resolutions submitted to TMA by the Tennessee Association for Home Health. One resolution calls for TAHH to form an interprofessional committee to work with TMA. The second resolution calls for physicians to approve only those home health services which are medically indicated; to approve home health services only to those patients to whom the physician provides care; and recognizes the physician as the primary determinant of home health care services.
- —Recognized Drs. John R. Nelson, Jr., Hugh Francis, Jr., Thurman L. Pedigo, Thomas K. Ballard, and William F. Buchner for their service to TMA as members of the Board of Trustees.
- —Elected Drs. Arden J. Butler, Jr., chairman of the Board of Trustees; Paul R. Stumb, vice-chairman; Jack Butterworth, Jr., secretary-treasurer; and Mr. L. Hadley Williams, assistant secretary-treasurer.
- —Appointed Dr. Thurman L. Pedigo, McMinnville, chairman of the IPA Implementation Task Force.

Third Quarter Meeting—July 13, 1986

The Board:

—Approved the general organizational structure of a statewide TMA-IPA as recommended by the IPA Task Force.

—Received a report from Dr. Sanders that the Joint Underwriting Association Board had voted to return $4.5 million to participating physicians in 1986.

—Approved the appointment of an ad hoc committee to work with Medicaid on the issue of Medicaid uniform reimbursement, as directed by Resolution No. 6-86, adopted by the TMA House of Delegates in April 1986.

—Received a report on the status of plans for TMA cosponsorship of the Southeastern Conference on Drug Abuse, Sept. 24-26, 1986 in Nashville.

—Voted to increase the price for TMA's contract evaluation service to $225 for the first person or group requesting the evaluation. All subsequent evaluations will be available for a fee of $25.

—Approved TMA cosponsorship of a Geriatrics Symposium, to be held in conjunction with a regional meeting of the American Medical Directors Association in December 1986 in Nashville.

—Received a report from TMA Auxiliary president, Mrs. Joanne Edmonson, that Tennessee received two membership awards and five AMA-ERF awards.

—Received a report from Dr. Lewis that SVMIC has been given an A+ rating by A.M. Best, Inc.

—Received a report that HCFA has awarded the PRO contract to the Mid-South Foundation for Medical Care. HCFA is interested in regionalizing, and in the future Tennessee may be bidding on contracts in other states.

—Received a report that IMPACT has a total membership of 907 (701 sustaining members, 111 corporate contributors, 95 auxilians).

—Approved the use of Tharp Award funds for developing a computerized records system for the Impaired Physician Program.

—Received a report from TMA Executive Director L. Hadley Williams on the formation of a statewide coalition for tort reform. Each participating organization has been asked to contribute $500 for membership. Individual memberships will be $100. The Board approved $500 for TMA membership in the Tennessee Association for Civil Justice Reform.

—Voted to support the Tennessee Society of Obstetrics and Gynecology's opposition to proposed regulation of state perinatal centers.

—Voted to appoint the physicians serving on the Medicaid Medical Care Advisory Committee as ex-officio members of the TMA Governmental Medical Services Committee.

—Voted to support the AMA pilot study designed to place physicians in schools as volunteer instructors in drug abuse prevention programs.

—Appointed Dr. Phillip Pedigo, Memphis, as TMA's representative to the Tennessee Non-Smoking Coalition.

—Voted to submit for consideration of appointment to the Board of Medical Examiners the name of Dr. John Dorian, Memphis.

—Appointed Dr. William P. Stallworth, Knoxville, as a member of the TMA Committee on Hospitals. Appointed Drs. Kimball Maull of Knoxville, John Morris, Jr., of Nashville, and Riley Jones of Memphis as members of the TMA Emergency Medical Services Committee.

Fourth Quarter Meeting—October 12, 1986

The Board:

—Received a report from Dr. Thurman L. Pedigo that the TMA-IPA has a total of 1,088 members to date. The TMA-IPA Task Force has entered into a contractual arrangement with Bay State HMO Management Company of Boston to conduct a feasibility study. The Board voted to authorize the TMA-IPA Steering Committee to incorporate the IPA.

—Received a report from TMA Staff Attorney Jere Sue Adams that the Tennessee Association for Civil Justice Reform (of which TMA is a member) has hired a public relations firm to develop strategies for coalition efforts. A total of 301 individual TMA physician members have joined the coalition.

—Received a report from TMA Executive Assistant Mark Greene that the Governor's Task Force on Tort Reform had concluded its hearings and would begin work on its final report later in the month. TMA leaders provided well-organized testimony in each hearing location, as did SVMIC.

—Received a report from Dr. Ted Galyon on the initial meeting of the TMA Hospital Medical Staff Section Committee. The committee has adopted governing principles for approval by the TMA Board. The Board voted to appoint Dr. John R. Nelson, Jr. of Knoxville as chairman of the Hospital Medical Staff Section until the initial Section meeting on March 1, 1987, in Nashville.

—Received a report from Dr. Hays Mitchell on his testimony at a public hearing conducted by the State Perinatal Advisory Committee. Dr. Mitchell reported that no testimony was given in support of the proposed perinatal regulations.

—Received a report from TMA Executive Assistant John Grant that a total of 14 medical specialty society programs have been approved for Category 1 CME credit in conjunction with the 152nd annual meeting of TMA, April 8-11, 1987.

—Received a report from TMA Auxiliary president, Mrs. Allen Edmonson, on Auxiliary activities.

—Received a report from Dr. James Royal that SVMIC's Loss Prevention Seminars would be limited to five or six during 1987 and each would consist of one-half day sessions. A $2-million dividend to stockholders has been declared. Dr. Lewis noted that the number of claims for the year has decreased but has increased in severity.

—Received a report from Dr. Clarence Sanders that Mid-South Foundation for Medical Care has signed a new contract with HCFA and that precertification has been reduced to five procedures. He urged physicians to continue to use the published guidelines to avoid difficulties and noted that readmissions will have more review than in the past.

—Received a report from Dr. Charles Thorne, chairman of the TMA Impaired Physician Committee, requesting renewal of the medical director's contract at the current salary level, including a provision for vacation time, backup or "on-call" services during the director's leave time, and support of AMA's request of participation in an impaired physician system pilot project. The Board voted to renew Dr. David Dodd's contract at the same salary level with a provision to include four weeks' vacation. The Board voted to refer to the Finance Committee the Impaired Physician Committee's request for providing "on-call" services during Dr. Dodd's leave time. The Board voted to approve participation in the multistate data base on impaired physicians.

First Quarter Meeting—January 17-18, 1987

The Board:

—Voted to approve the Impaired Physician Committee recommendation that the physician covering the vacation periods of the medical director of the Impaired Physician Program be on call 24 hours per day.

—Received a report from the staff that TMA and individual physicians had made contributions to the Tennessee Association for Civil Justice Reform totaling $44,245, and that proposed legislation was being drafted for introduction in the Tennessee General Assembly.

—Received a report from staff that a not-for-profit charter had been filed and approved by the Secretary of State's office in the name of Tennessee Physicians Practice Association, Inc. and that all funds collected by TMA on behalf of the IPA had been transferred to the new corporation prior to Dec. 31, 1986.

—Received a report from staff that the State Board of Medical Examiners will develop a new testing instrument for x-ray examinations that is more compatible with the approved curriculum.

—Approved the ad hoc committee to study TMA dues categories' recommendation that physicians taking leave of absence for military service, public health service, maternity leave, etc., be granted dues exempt status for the duration of their absence upon written application to TMA. The Board also approved: (1) the committee's recommendation that physicians less than 70 years of age who work less than 20 hours per week be permitted to pay one-half dues if certified to TMA by their component medical society on an annual basis; (2) the recommendation that physicians entering practice for the first time be allowed to pay one-half dues for the first two years. If a physician enters practice for the first time and is certified to TMA after July 1, one-fourth of the dues will be payable upon certification and one-half dues will be payable for the two succeeding years; (3) a recommendation that two-physician families continue to pay full dues so long as both physicians are working fulltime. If one physician works less than full time, then the appropriate category would apply.*

—Nominated and approved physicians to serve on each of the standing and special committees of TMA. All appointments will become effective at the conclusion of the TMA annual meeting in April. A list of appointees are on file in the TMA office.

—Reappointed Drs. Billy J. Allen of Ooltewah and Allen S. Boyd, Jr. of Memphis to three-year terms on the Board of Directors of the TMA Student Education Fund.

—Appointed Dr. Rod Kirkpatrick of Memphis to replace Dr. Rex A. Amonette of Memphis as the IMPACT Board member representing the ninth congressional district for a one-year term; all other IMPACT Board members were reappointed for one-year terms.

—Reappointed Drs. C. Eugene Jabbour of Memphis and John B. Thomison of Nashville to the Board of the Tennessee Medical Foundation. Appointed the following to the TMA Nominating Committee: representing East Tennessee—Drs. Duane C. Budd of Johnson City, Pete S. Soteres of Chattanooga, Kent Farris of Knoxville; representing Middle Tennessee—Drs. William Pedigo of Clarksville, James W. Hays of Nashville, Charles E. Jordan, III of Cookeville; representing West Tennessee—Drs. James W. Shore of Martin, Charles White of Lexington, Phillip A. Pedigo of Memphis.

—Voted to introduce a resolution at the 152nd annual meeting which would encourage TMA members to authorize medical exemption for seat-belt use only in those instances where safety considerations afforded by seat-belt usage are outweighed by the inappropriateness of restraints given the physical handicap.

—Heard a report from Dr. Kenneth J. Phelps, Jr. of Lewisburg, chairman of the Board for the Mid-South Foundation for Medical Care. Dr. Phelps discussed problems being encountered as a result of the new PRO contract issued to the Foundation by HCFA.

—Accepted a report from TMAA president, Mrs. Allen Edmonson, who outlined for the Board the program for the Teen Health Workshop to be conducted in Memphis on March 5, 1987 at Memphis State University.

—Voted to award the 1987 Distinguished Service Awards to Drs. Howard Foreman of Nashville, James W. Hays of Nashville, and David H. Turner of Chattanooga.

—Voted to reaffirm the policy concerning a continuation of the four levels of nurse training in Tennessee presently contained in Resolution No. 5-80 and to reintroduce the resolution at the next session of the TMA House of Delegates.

—Voted to go on record in support of the Tennessee Hospital Association's plan to fund indigent care in Tennessee.

—Appointed Drs. Allen M. Bisno of Memphis, Steven S. Hawkins of Chattanooga, and William Schaffner, II of Nashville to an ad hoc committee to study issues regarding the disposal of infectious wastes.

—Reappointed Mr. Charles L. Cornelius, Jr., as TMA legal counsel and Mr. Bob Bellenfant as TMA auditor for 1987.

—Approved the unaudited 1986 financial statement showing receipts of $1,168,329.41 and expenditures of $1,163,392.09.

—Approved the 1987 budget with projected income of $1,433,000 and expenditures of $1,350,250.

These are just some of the areas that we have touched on. Others will be listed on the pages that follow. On behalf of the Board of Trustees, I would like to thank Hadley Williams, Don Alexander, and the TMA staff for the very dedicated and competent work that they do. These people truly have our interest at heart and go above and beyond the call of duty frequently in their jobs. I would single out one other member of the administrative staff for special comment. Ron Gant has held basically two jobs for most of the year and done them both well. He traveled from one end of the state to the other working with IPA, and has done and is doing an excellent job in the legislative area. Mr. Gant will be leaving TMA's employ following the current session of the Tennessee Legislature and will go to work full time for Tennessee Physicians Practice Association, Inc.

It has been a privilege and a pleasure to serve as chairman of your Board of Trustees for the past year. The chance to work with this many people of this high caliber has made it an exhilarating year.

***The House of Delegates adopted the recommendations of Reference Committee C, as presented in the Report of the Board of Trustees, pertaining to the changes in dues categories. These changes will be effective beginning 1988.**

REFERENCE COMMITTEE C—*reviewed the report of the Board of Trustees, expressed its appreciation to Dr. Butler and the Board for their accomplishments on behalf of the Association during the past year, and recommended that the report be filed.*

Report of the Judicial Council

Howard L. Salyer, M.D., *Chairman*

The Judicial Council of the Tennessee Medical Association held only one formal business meeting this past year. The House of Delegates in 1986 referred two resolutions to the Judicial Council for its review and opinion. Resolution No. 18-86, entitled Physician Orders for Terminally Ill Patients in Nursing Homes, and Resolution No. 19-86, entitled Patient Autonomy were carefully considered with consultation from the American Medical Association.

It was the opinion of the Judicial Council that the members of the Association did need guidelines as related to these two resolutions. After these resolutions were referred to the Council last April, the American Medical Association, through its Council on Ethical and Judicial Affairs, adopted guidelines that directly relate to the problems presented in Resolutions No. 18-86 and No. 19-86. Attached to this report, you will find excerpts from the Current Opinions of the Council on Ethical and Judicial Affairs of the American Medical Association 1986. Each member of the Association is encouraged to become familiar with these sections as they relate to withholding or withdrawing life-prolonging medical treatment and patients' preferences as they relate to these issues.

The Council also considered requests for opinions regarding fee splitting and contracts that tend to encourage unethical practice by physicians. The Council felt these issues should best be discussed in resolution form, which you will find before you in Resolutions No. 4-87 and No. 5-87. The Judicial Council wholeheartedly endorses these resolutions and encourages adoption by this House of Delegates.

The only other matter that came to the attention of the Judicial Council was from a member relating to suspension from his component medical society. This appeal was thoroughly researched, discussed, and the unanimous opinion of the Council was to remand to the component society.

Several other items came to the attention of the Judicial Council and were referred to component societies for resolution. As usual, our component medical societies do an excellent job of interpreting and handling ethical issues that come before them regarding their membership.

As chairman of the Judicial Council, I wish to express my appreciation for the outstanding service that has been rendered this year by members of the Council and thank them for their cooperativeness and diligence in their deliberations.

REFERENCE COMMITTEE C—*reviewed the report of the Judicial Council, expressed its appreciation to Dr. Salyer and members of the Council for their continued excellent work during the past year, and recommended that the report be filed.*

ATTACHMENT 1 TO REPORT OF THE JUDICIAL COUNCIL

Current Opinions of the Council on Ethical and Judicial Affairs of the American Medical Association

2.18 Withholding or Withdrawing Life-Prolonging Medical Treatment

The social commitment of the physician is to sustain life and relieve suffering. Where the performance of one duty conflicts with the other, the choice of the patient, or his family or legal representative if the patient is incompetent to act in his own behalf, should prevail. In the absence of the patient's choice or an authorized proxy, the physician must act in the best interest of the patient.

For humane reasons, with informed consent, a physician may do what is medically necessary to alleviate severe pain, or cease or omit treatment to permit a terminally ill patient whose death is imminent to die. However, he should not intentionally cause death. In deciding whether the administration of potentially life-prolonging medical treatment is in the best interest of the patient who is incompetent to act in his own behalf, the physician should determine what the possibility is for extending life under humane and comfortable conditions and what are the prior expressed wishes of the patient and attitudes of the family or those who have responsibility for the custody of the patient.

Even if death is not imminent but a patient's coma is beyond doubt irreversible and there are adequate safeguards to confirm the accuracy of the diagnosis and with the concurrence of those who have responsibility for the care of the patient, it is not unethical to discontinue all means of life-prolonging medical treatment.

Life-prolonging medical treatment includes medication and artificially or technologically supplied respiration, nutrition or hydration. In treating a terminally ill or irreversibly comatose patient, the physician should determine whether the benefits of treatment outweigh its burdens. At all times, the dignity of the patient should be maintained. (I,III,IV,V)

2.19 Withholding or Withdrawing Life-Prolonging Medical Treatment—Patients' Preferences

A competent, adult patient may, in advance, formulate and provide a valid consent to the withholding or withdrawal of life-support systems in the event that injury or illness renders that individual incompetent to make such a decision. The preference of the individual should prevail when determining whether extraordinary life-prolonging measures should be undertaken in the event of terminal illness. Unless it is clearly established that the patient is terminally ill or irreversibly comatose, a physician should not be deterred from appropriately aggressive treatment of a patient. (I,III,IV,V)

Report of the Secretary-Treasurer

Jack Butterworth, Jr., M.D.

The annual audit for the fiscal (and calendar) year ending December 31, 1986 has been completed and is available for review. The customary examination of Association records was conducted by Bellenfant & Miles, P.C., Certified Public Accountants.

The attached financial reports have been extracted from the complete audit. They show the revenue and expenditures during 1986 as well as the assets, liabilities, and fund balance at the end of the year. A small deficit of just under $6,000 reduced the total equity to $1,868,327, including $244,713 in fixed assets. The operating reserve, based upon projected 1987 expenditures, dropped to about 14 months.

We narrowly escaped operating deficits during the preceding two years, by $14,000 in 1985 and by $20,000 in 1984. Without the infusion of funds from the recently formed TMA Physician Services, Inc., a deficit of more than $70,000 would have been recorded. Investment income, a major factor in previous years in avoiding deficits, dropped to $142,744 in 1986, down from the $165,397 amount the previous year. The continued decline of interest rates will lower investment income even more in 1987.

The modest dues increase approved by the House last year will enable TMA to avoid a second consecutive deficit. The current $220 annual TMA dues continues to be among the lowest in the country with only three states—Oklahoma, Delaware, and Nevada—having lower state association dues in 1987.

The Association remains in sound financial condition. Prudent investment management and operating policies are followed.

TENNESSEE MEDICAL ASSOCIATION STATEMENT OF REVENUE, EXPENSES AND FUND BALANCE

	Year ended December 31	
	1986	**1985**
REVENUE		
Membership Dues (net to *Journal*)	$ 742,591	$ 733,655
Annual Meeting—Exhibits	37,245	26,640
Annual Meeting—Tickets	16,402	7,105
Investment Income	142,744	165,397
AMA Collection Fees	8,813	7,408
Impaired Physician Grant	84,502	12,951
Other Income	1,650	
Total Revenue	$1,033,947	$ 953,156
EXPENSES		
Administrative	$ 437,885	$ 464,979
Administrative Support & Services	19,930	19,161
Travel—Staff	30,898	28,284
Officers	62,197	55,290
Impaired Physician Program	156,570	83,660
Committee Expense	16,292	22,142
Legislative Committee	38,456	33,546
Student Education Fund	121,838	112,004
Other Organizations	9,780	8,980
Annual Meeting	49,742	34,080
Taxes	28,288	27,956
Headquarters Expense	26,888	24,060
Depreciation Expense	24,797	24,147
Contingencies	1,553	750
Total Expenses	$1,025,114	$ 939,039
Excess of Revenue Over Expenses Before *Journal*	8,833	14,117
Excess *Journal* Expenses	(14,587)	(12)
Excess of Revenue Over (Under) Expenses	(5,754)	14,105
FUND BALANCE		
Beginning	1,874,081	1,859,976
Ending	$1,868,327	$1,874,081

TENNESSEE MEDICAL ASSOCIATION BALANCE SHEET

	Year ended December 31	
	1986	**1985**
ASSETS		
Cash	$ 74,871	$ 173,932
Investments	1,955,314	1,689,547
Interfund Notes Receivable	89,700	143,900
Accrued Interest Receivable	78,020	79,285
Other Receivables	3,694	4,232
Total Current Assets	$2,201,599	$2,090,896
Real Estate and Equipment (net)	244,713	256,405
Total Assets	$2,446,312	$2,347,301
LIABILITIES		
Accounts Payable and Accrued Expenses	$ 12,443	$ 13,450
Dues Collection Escrow	565,542	456,208
Deferred Revenue		3,562
Total Current Liabilities	577,985	473,220
FUND BALANCE	1,868,327	1,874,081
Total Liabilities and Fund Balance	$2,446,312	$2,347,301

TENNESSEE MEDICAL ASSOCIATION JOURNAL INCOME AND EXPENSES
Year Ended December 31, 1986

	Total	**Readership**	**Advertising**
INCOME			
Allocation of dues	$ 77,424	$77,424	$ —
Advertising	57,468	—	57,468
Subscriptions	2,140	2,140	—
Total Income	$137,032	$79,564	$57,468
EXPENSES			
Printing and Distribution	$97,921	$65,764	$32,157
Editor and Board	3,095	3,095	—
Clerical Assistance	600	600	—
Clipping Services	1,718	1,718	—
Salaries	20,500	10,250	10,250
Employees Insurance	2,122	1,061	1,061
Taxes	1,535	768	767
Supplies	—	—	—
Staff Travel	543	271	272
Overhead	23,585	15,723	7,862
Total Expenses	$151,619	$99,250	$52,369
JOURNAL INCOME (Loss)	($ 14,587)	($19,686)	($ 5,099)

REFERENCE COMMITTEE C—*reviewed the report of the secretary-treasurer, thanked Dr. Butterworth for the good work he has done during the past year, noted that it is pleased with the financial management of the Association, and recommended that the report be filed.*

Report of the Executive Director

Mr. L. Hadley Williams

My report to the House of Delegates deals primarily with administrative matters of the Association. It is difficult, however, to try and convey the true picture of the amount of work centered around the headquarters office and staff. Many of the reports being received by the House today are a reflection of the creativity of the staff and do more to indicate the work of the Association during the past 12 months than any summary I might present. I believe we have a very responsive and talented staff and that they represent the physician members in a most complimentary manner.

Responding to the needs and desires of almost 6,000 members is a most difficult task. The annual session of this House is a revealing time of stock-taking and the method by which TMA can analyze its situation and evaluate its position on major policy matters. This House, as the TMA governing body, must decide policy which will best represent the state's medical profession and ultimately influence the entire medical and health industry. Responding to the needs of the membership is no easy task, but it is one that is vital to the effective, productive operation of the TMA.

I am pleased to report that the financial condition of TMA is sound. I recommend the Secretary-Treasurer's Report to you as a detailed accounting of assets and liabilities and an overall picture of the financial structure.

In order to support the Association's increasing level of activities and programs, the Board of Trustees recommended and the House of Delegates approved a $50 dues increase effective January 1987. The new dues total of $220 per year ranks Tennessee well below the national mean of $273 for state associations. It is interesting to note that even with the dues increase, only three states have dues lower than Tennessee's $220.

For the past two to three years, the cost of medical care was the predominant theme. Last year, cost of care criticism seemed to subside as the professional liability insurance crisis issue again reared its ugly head. However, we see strong signs that the health costs issue appears to be rising again. We are seeing a restructuring of the manner in which medical services are contracted and paid for. The distinction between hospitals and insurers is blurring. Medicine, traditionally based on understanding and trust between physician and patient, is now big business. Employers are beginning to assume the same posture as government, thereby demanding that they play a greater role in deciding how, when and where care is delivered, and how much they will pay for services. Medicine must find ways to respond to this situation to assure that the patient is given the highest quality of care available, and the employer recognizes that his employees are receiving maximum value for the premium dollar.

A strong and unified voice is vital and is needed more today than ever before. Only through a united voice on the part of the county societies, TMA and the American Medical Association can the challenges of the FTC, HCFA, cost containment, PROs, and others be effectively addressed. Physicians should look on their local, state, and national organizations as an investment—of time and money. But if you don't make an investment, you can't expect any dividends.

I would like to commend the staff for their dedication and devotion to duty during the past year, and I would like to take this opportunity to thank all of the officers and members of the Board for the many hours they have contributed on the part of the profession and for the leadership that they have provided TMA during the past 12 months. Their task is a thankless and oftentimes an unrewarding one. Dr. James Royal has unselfishly provided the necessary direction and leadership and I know Dr. Ted Galyon will take up the torch and will continue to render the strong leadership the Tennessee Medical Association will require as we move ahead.

TMA MEMBERSHIP REPORT
As of December 31, 1986

	1986	1985	1984	1983	1982
Dues Paying Active Members	4,911	4,834	4,772	4,714	4,616
Dues Paying Resident Members	71	79	71	79	87
Dues Exempt Members	830	787	751	761	611
Veteran Status . . .502					
Military, Disabled and Retired 194					
Student 134					
TOTAL	5,812	5,700	5,594	5,554	5,314
Deaths	41	34	51	43	39

AMA members from Tennessee Medical Association:

Dues Paying	3,706
Dues Exempt	566
Direct Members	1,766
TOTAL AMA MEMBERS	6,038

75% of dues paying TMA members are dues paying AMA members

REFERENCE COMMITTEE C—*reviewed the report of the executive director, expressed the appreciation of the TMA membership to Mr. Williams for his excellent job, and recommended that the report be filed.*

Committee Reports

The following standing and special committees made annual reports to the House of Delegates:

—Committee on Scientific Affairs
—Committee on Legislation
—Committee on Governmental Medical Services
—Committee on Hospitals
—Peer Review Committee
—Committee on Communications and Public Service
—Interprofessional Liaison Committee
—Committee on Continuing Medical Education
—Impaired Physician Peer Review Committee
—Committee on Blood Banks and Medical Laboratories
—Committee on Rural Health
—Committee on Emergency Medical Services
—Advisory Committee to TMA Auxiliary
—Committee on Medicine and Religion
—Committee on Maternal and Child Care
—Primary Care Liaison Committee
—Geriatrics Committee
—Committee on Medical Practice

James T. Galyon

Cost Containment, At What Cost?

The TMA Annual Meeting had many facets, but to this observer much of the activity and many of the resolutions brought to the House of Delegates dealt with issues caused by efforts at cost containment.

We are continually told by the press that "the spiraling cost of health care" has to be contained. This is the same press that raised its home delivery charge by 10% this year. We are told the same thing by a Congress that voted itself a large salary increase this year and by a state legislature that duplicated that act. It seems only medical costs need containment.

At our House of Delegates we dealt with resolutions addressing admissions on the same day of surgery, PRO denials and sanctions, Blue Cross denial of payments for certain tests, contracts that place the physician in unethical positions, combined payments of global fees to RAP physicians and the hospitals where they work, generic prescribing and abuses, PPOs that fail to mention that their institutions and doctors are "preferred" only because they discount their services and render care at a "contained cost," and finally with insurance policies that appear to offer adequate coverage to the unsuspecting public, but which actually erect obstacles to health care all in the name of "cost containment."

Somewhere in this mandate to control the cost of health care several important factors have been left behind. There must be enough money in the equation to fund postgraduate education and medical research. Indigent medical care can no longer be financed by cost shifting if there is little money left to shift. And certainly the rush to contain cost has slighted quality of care by limiting hospitalization to the extent that the patient's health may be jeopardized. Generic drugs, lacking the assurance of comparable bioequivalency and bioavailability, are being substituted at geographically remote pharmacies only to reduce costs.

Perhaps the whole idea of "spiraling health care costs" is out of proportion. The usual figure quoted is about 9% of the gross national product. Lumped into this figure are the expenses of domiciliary care for the elderly in nursing homes and feeding infants and children in some programs. While these are laudable social activities, they are not medical expenses. This inflated figure almost reaches the cost of recreation.

We have a responsibility to wisely utilize health resources, but we have a greater responsibility to render quality care. This may require us to spend some of our time acting as advocates of our patients with utilization review boards, PROs, and insurance companies.

Cost should reflect the value of the service received. I believe the cost of health care, while expensive, produces an enormously valuable product—the best health system in the world.

James T. Galyon, M.D.

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JUNE, 1987

editorials

Gimme a Light

It is at once a mark of the perversity and the genius of man not to leave well enough alone. Though God was content to rest from the labors of His grand design on the seventh day, which has persisted until now, man has always felt obligated to muddy the waters by pushing out into new frontiers, perhaps out of a restlessness conferred on him in Eden by the snake. It led him to Babel and beyond, and continues to manifest itself. It gets him to dabbling in drugs; not content with alcohol, man has made his way through opium to snow to coke and crack and on to "designer drugs," each with its in days and out, and its own peculiar dangers. Wagner enlarged on Beethoven, who had done the same to Haydn, and each was vilified for it. Schoenberg simply extended the process. I personally think he went too far, but many of the contemporaries of Beethoven and Wagner thought the same about them. As the hat becomes old and stodgy, it is cast aside by the avant garde.

I suppose there is no rule that says this piece of paper, and therefore books, should take the shape of an upright rectangle, but regardless of size, that has been the custom since long before the advent of moveable type. Because the eye scans a narrow column more easily than a broad one, this and other publications not only are vertical, but even put two or three columns on a page to make what the eye must scan even narrower.

Except for man and trees and a few other upright creatures and some of man's concoctions, the sweep of nature is horizontal and not vertical, and so artists' canvases have extended more often that way. It was only natural, then, that sooner or later books displaying their creations would take that form. They did, once reproduction became satisfying enough to make coffee table editions salable. Because those monstrosities would not fit into bookcases or anything else designed to hold books, the coffee table is where they landed.

That may have been a result of genius, but it has to be out of nothing but perversity that would put a working document in such a format, since it will fit into no receptacle made by either God or man (except a trash bin), and it is not fittin' for any coffee table, either. I am speaking now particularly of the long awaited Health Policy Agenda for the American People (HPAFTAP). Not only does it defy storage, it also frustrates any usage at all. Being limp, and not rigid, it is impossible to hold, or do anything useful with.

I have an idea who is responsible for that miscarriage; it comes from some of the same sort as those who find fault with the design of this *Journal*, and who are forever trying to get us to change it, at great cost. Though the *Journal* won awards for design in 1980 and 1983, we haven't kept up with the times. We ain't chic: our format hasn't changed. Neither have the individuals giving the awards, of course; only the fads have.

I haven't heard any complaints about the format of the *Journal* from our constituency—only from the judges. (If you have any complaints, please communicate them to me. I am not too old to change, given proper reason, and from the proper people, who are the members of this Association; but remember, it will cost you money. We don't think it is cost effective—and that *is* chic.) I propose eventually to write a critique of the HPAFTAP for you (pronounced H'Paftap—I just now made that acronym up, in line with all the others lying about; the "haitch" is an aspirate, and the "pee" is silent, as in pswimming, so that it sounds like "half tap," after beer—as an old fud, I stick with that, instead of more chic substances). That is, I will if I can ever figure out a way to hold their report.

I believe I've worked that one about as much as I can. Gimme a light—and *I* don't mean Bud Light; it's closer to water than beer. I mean—like, man—Send the Light! Why would they put such a very important document as HPAFTAP in such a ridiculous container? It blows the mind.

See you around.

J.B.T.

Heah Come de Judge

I got this kid, see, and I don't have a house, and so I don't have a room for him to live in. And so I find this lady, see, who does have one, and so I rent it from her. I say, now when I got to take him home I may have to knock a hole in the wall to get him out. She says that's okay—gimme $10,000. And so I do—give her the ten grand and knock a hole in the wall and take him home. But then she says, you tore up my house—gimme the kid back. I say nothin' doin'—I got a contract. I say the kid goes with me. So does the judge.

What Judge Harvey Sorkow said was in essence that it made no difference that Mr. Stern's kid was only a few microns long when he rented Mrs. Whitehead's room—er—*womb*. Each had a constitutional right over their respective parts, and the rental agreement was binding, and the kid goes with Mr. Stern. Mrs. Whitehead will appeal the decision.

Judge Sorkow clearly had a sticky wicket. Divorce courts have almost always, barring some maternal defect, awarded child custody to the mother, because the relationship of father and mother in procreation is something like that of the hen and the hog at breakfast: the hen is involved, to be sure, but the hog is committed. Daddy's role is over pretty quickly, and wasn't all that much of a chore, either. Mama's goes on for a while, and terminates at best in turmoil. Nevertheless, a bargain's a bargain, said Judge Sorkow. The appeal will likely go on long enough that little Miss Stern, nee Whitehead, will carry memories of it to her grave.

There are, as has become apparent during the past few weeks, a variety of opinions about both the legality and morality of surrogate motherhood. Nevertheless, it seems to me that given the precedent of *Roe vs Wade,* Judge Sorkow had no alternative but to rule as he did. Mr. and Mrs. Stern wanted a baby, and normal means toward that end had been unsuccessful. They found a needy young woman who was willing to rent her womb and proceeded to take her up on it. During the pregnancy, with its inconveniences and woes (and I suspect especially during parturition) Mrs. Whitehead repented her decision, and welched on her agreement. The Judge said it won't wash. It is not the same as child selling, he said. She was selling services: rental property.

Society and all its components have always wanted to have it both ways. Women have always sold their services to willing buyers, and society, after making it illegal, has gone about in a rather desultory fashion enforcing its statutes. One obstacle to buying was the consequences, which were mitigated, if not obliterated, by penicillin and the Supreme Court. Once a woman could evacuate her womb at her pleasure, it is only natural that she be allowed to fill it in the same way—including rental, if she considered that to her advantage.

The bar sinister is heraldry's acknowledgment to the fruits of illicit love. Society legitimatized illicit love, though not its fruits, and in fact, if you believe the soaps, encourages it. Furthermore, it is no longer requisite to raise its consummation; you simply flush the bastard out. If you have extra space, why not rent it out? Of course, it will prove inconvenient for a while, which Mrs. Whitehead apparently failed to appreciate. As we used to say in the Army, TS.

If the Supreme Court wishes not to speak with forked tongue, it will have either to uphold Judge Sorkow or repudiate *Roe vs Wade*. The problem arose when society turned its back on the monogamous relationship. It is seldom possible to have your cake and eat it. Fill an unwed womb, and you have a problem, whatever either society or the Judge says.

J.B.T.

A Tale of Two Women

Time passes and memories fade; even those of modern times, which should be kept intact through documentation on celluloid, are suspect, as those are often edited to suit the whims or political philosophies of their proprietors, dredging up as they do lots of detritus best forgotten and suppressing sometimes momentous happenings. Much of the replay is long on political activism and short on verity, and only proves that facts, like statistics, can be rearranged to perpetuate lies and damned lies. So you needn't depend on the TV documentaries for your history. Not that the history books are any better, and memoirs are notoriously slanted—and why not?—which means that history as lived is very difficult, if not impossible, to get at.

But not always. The movies—and by that I mean the feature films—admittedly give a biased view of life as viewed from Hollywood (or Hollywood abroad), which like the historical novel is mostly confection. But the airing of the "golden oldies" does do one thing: it keeps alive some burning infatuations—which is usually translated "nostalgia."

Mention the name Leslie Howard, and it will undoubtedly call to mind the vapid Ashley Wilkes of *Gone with the Wind*—affectionately shortened by my generation to *GWTW.* Most folks forget that Leslie Howard had already been around a while, which was why he got the role in the first place, and that he indeed preceded Rex Harrison as Professor Henry Higgins—not in *My Fair Lady (MFL),* but in George Bernard Shaw's *Pygmalion,* the daddy (or mother, if you prefer) of *MFL.* He was also a great Hamlet—in short, a fine actor.

That is not what I remember Mr. Howard for, though. I remember him because he introduced as his co-star in a film entitled *Intermezzo* (not a great movie, by the way) a young Swedish actress, one already well known in Sweden but just discovered by Hollywood, named Ingrid Bergman. I was in high school, and I was in love with her then and I still am; the reruns on the tube see to that. Miss Bergman starred in many fine films, such as *For Whom the Bell Tolls* and *Going My Way,* but the one for which she is best remembered, of course, is one of the great all time classics with Humphrey Bogart, Claude Raines, and Paul Henreid—*Casablanca.* She refused the demands of Hollywood that she change her teeth, her hair, and her name; she offered to go back to Sweden, where they liked her as she was. We can all be grateful that she charmed Hollywood, too.

When the marriage of Ingrid Bergman with Dr. Peter Lindstrom crumbled in favor of Italian movie director Roberto Rossellini, the busybodies, led by those arbiters of Hollywood virtue Louella Parsons and Hedda Hopper, who never liked Miss Bergman because they were not among her favorites, either, slopped a large blot on her escucheon, and she was driven from our noble shores in disgrace (but not by me). After sleeping around and getting divorced came into vogue (outside of Hollywood, that is, where it never was out) Bergman returned in triumph and made many more films, winning some Academy Awards, and also starring in some Broadway plays. Folks learned from that (or some did) that one had better not stare too hard at the feet of even the brightest of idols—which they should have known already.

A trooper to the end, while she was dying of cancer Ingrid Bergman made her last film about another bright idol whose feet came as close to matching the rest of her as just about anybody who ever was. In *A Woman Called Golda,* Bergman portrayed the monumental Golda Meier, who has been called the Mother of Israel. Considering her uncanny powers of persuasion, one has to wonder whether without her Israel might even not exist today as a nation. For her whole life, Golda Meier was driven by a single-minded devotion to the idea of a Jewish homeland, and with the possible exception of David Ben-Gurion, did more than anyone else to make it a reality.

The obvious similarities between these two remarkable women could not have escaped Miss Bergman, who came literally from her deathbed to make this television feature. Both were thoroughly professional individuals totally devoted to their craft. They were wholly candid and open with everyone, and always true first to themselves. Being as they were brought them into conflict with their feminine instincts of motherhood. Four marriages died on the block of professionalism—three for Bergman and one for Golda—and six children suffered deprivation. Golda's only expressed regret about her life was her failure to make a home for her husband and children.

Except where physical strength is required, and in some situations not even then, there is nothing a man can do that a woman cannot do as well, even though it might be done differently, given what appear to be inherent differences in their respective thought processes. The converse, however, does not apply in one situation, the importance of which is paramount. Men cannot bear children. Not only that, regardless of what the feminists would have you believe, men cannot make a home. As with every generalization, there are, of course, exceptions (except for the one about bearing children), but generally speaking that is so.

If it is chauvinistic and old-fashioned to believe that a woman's place is in the home, then I have to confess to being both, at least where children are involved. If a woman is to have children, then she ought to stay with them while they are young. Surrogate mothers are never the same. I am not speaking now of surrogate wombs. Somehow the definition of motherhood has become corrupted to speak of motherhood as if it were confined to the process of procreation. That is only where motherhood starts. Children need a mother for more things than just to bear them. It is at their mother's knee (natural or surrogate) that children learn whatever graces they will carry with them through life; the first four years have been found to be critical.

Mine is not a popular notion these days, nor in fact has it ever been. It is not even popular with me, since if that course were followed I would have been deprived of a great many associations that I have valued greatly in my professional life. Nevertheless, I am persuaded that Western Civilization is crumbling for want of homes, and, feminist observations to the contrary notwithstanding, it is the mother who makes the home. I recognize that there are some abandoned or bereft families where the mother must work so the children can eat, but children—even very young ones—understand the difference. Those children are seldom the ones who drift off into the drug culture and so on; they usually work at making the home.

The judgment of the world, myself included, is that it would have been the poorer without Ingrid Bergman and Golda Meier as we knew them. But whether the world's estimation is the right one or not, even granting that Israel believes Golda was sent from God, and maybe she was; only God knows for sure. I do know I wouldn't touch that one; I've touched enough already.

J.B.T.

in memoriam

John O. House, age 67. Died April 12, 1987. Graduate of University of Tennessee College of Medicine. Member of Chattanooga-Hamilton County Medical Society.

W. Houston Price, age 75. Died April 13, 1987. Graduate of University of Tennessee College of Medicine. Member of Chattanooga-Hamilton County Medical Society.

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

CAMPBELL COUNTY MEDICAL SOCIETY
Scott R. Lillibridge, M.D., Jellico

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY
Brian A. Glass, M.D., Chattanooga
Lawrence S. Nagel, M.D., Chattanooga
Vinit D. Patil, M.D., Chattanooga
Ann Harris Rybolt, M.D., Chattanooga

CONSOLIDATED MEDICAL ASSEMBLY OF WEST TENNESSEE
David Fanning, M.D., Jackson

KNOXVILLE ACADEMY OF MEDICINE
James C. Denneny, III, M.D., Knoxville

MEMPHIS-SHELBY COUNTY MEDICAL SOCIETY
Lee Allen, M.D., Memphis
George William Bounds, III, M.D., Memphis
Ann Dail Brown, M.D., Memphis
Pamela Stephens Bullock, M.D., Memphis
Roger Stephen Cicala, M.D., Memphis
Jack Colbert Morgan, M.D., Memphis
Barry Ross Siegel, M.D., Memphis
John C. Turley, III, M.D., Memphis

(Students)

Don T. Brothers, Jr., Memphis
Richard Cape, Memphis
James W. Clogston, Memphis
Robert B. Davis, Memphis
John Endsley, Memphis
John B. Hagan, Memphis

NASHVILLE ACADEMY OF MEDICINE
Robert H. Alford, M.D., Nashville
Allan H. Bailey, M.D., Nashville
Benjamin F. Byrd, III, M.D., Nashville
Susan B. Campbell, M.D., Nashville
Richard Eugene Carlson, M.D., Madison
Nancy Elizabeth Chaney, M.D., Nashville
Bryce William Dixon, M.D., Nashville
Marc Scott D'Angelo, M.D., Nashville
Susan Brittingham Gregg, M.D., Nashville
John Philip Guillermin, M.D., Hermitage
David Edwin Hill, M.D., Nashville
Bennett Franklin Horton, M.D., Nashville
Robert J. Maciunas, M.D., Nashville
George S. Scoville, Jr., M.D., Nashville
Richard C. Tomichek, M.D., Nashville

personal news

William H. Edwards, M.D., of the Edwards-Eve Clinic in Nashville, has been appointed to a third three-year term as Councilor for the state of Tennessee for the Southeastern Surgical Congress. Dr. Edwards, an active member of the Congress since 1978, has also been elected to the Executive Committee for a two-year term.

James A. Greene, M.D., Knoxville, has been selected to represent the American Medical Association on the Joint Commission for the Accreditation of Hospitals' Homecare Technical and Professional Advisory Committee.

Jens David Henriksen, M.D., Chattanooga, has received the President's Committee on Employment of the Handicapped "Physician of the Year Award". Dr. Henriksen, who is in the field of rehabilitation medicine and is medical director of the Siskin Foundation, initiated the community effort to organize and expand the Chattanooga Area Governor's Committee on Employment of the Handicapped, and has made hundreds of the city's business leaders alert to the hiring of handicapped people.

Gerald I. Plitman, M.D. was honored posthumously with the Ralph O. Claypoole Sr. Memorial Award for distinguished achievements in the clinical practice of internal medicine. Dr. Plitman served as chief of hematology and general medicine at Kennedy Veterans Hospital in Memphis, and was professor of medicine and head of the Section of Medical Practice at the University of Tennessee Medical School, Memphis. He was also American College of Physicians governor for Tennessee and president of the Tennessee Society of Internal Medicine.

Kenneth S. Verheeck, M.D., Jackson, has been certified as a Diplomate of the American Board of Urology.

TMA Members Receive AMA Physician's Recognition Award

Thirty-four TMA members qualified for the AMA Physician's Recognition Award during March 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Paul S. Auerbach, M.D., Nashville
Robert F. Baker, M.D., Sparta
Lana S. Beavers, M.D., Shelbyville
Terry C. Borel, M.D., Johnson City
Reuben A. Bueno, M.D., Nashville
Sung J. Chung, M.D., Morristown
Robert E. L. Craig, M.D., Chattanooga
James H. Crenshaw, M.D., Humboldt
Avtar S. Dhaliwal, M.D., Johnson City
Samuel H. Dillard, Jr., M.D., Nashville
J. Vance Fentress, M.D., Pulaski
George S. Flinn, Jr., M.D., Memphis
Philip Goldhammer, M.D., Memphis
Charles G. Graves, M.D., Dunlap
Robert J. Gubler, M.D., Johnson City
Thomas E. Hanes, M.D., Madison
George L. Holmes, III, M.D., Nashville
David H. Horowitz, M.D., Nashville
Ronald J. Johnson, M.D., Memphis
Daniel P. Labrador, Jr., M.D., Chattanooga
James J. Madden, Jr., M.D., Brentwood
G. Patrick Maxwell, M.D., Nashville
David H. McConnell, M.D., Newport
Albert J. Mitchum, M.D., Clarksville
Peter A. Oliva, M.D., Johnson City
Ronald E. Overfield, M.D., Nashville
Harry L. Peeler, M.D., Selmer
Harold B. Reeder, M.D., Jackson
Robert A. G. Ricketson, M.D., Nashville
Billy G. Robbins, M.D., Halls
Lyle R. Smith, M.D., Kingsport
Montie E. Smith, Jr., M.D., Selmer
John M. Tanner, M.D., Nashville
Bruce I. Turner, M.D., Nashville

announcements

CALENDAR OF MEETINGS

NATIONAL

July 3-5	North Carolina Medical Society, 17th Annual Sports Medicine Symposium—Blockade Runner Hotel, Wrightsville Beach, N.C.
July 5-9	Health Physics Society—Hotel Utah/Salt Lake City Convention Center
July 10-12	Olympic Festival Congress on Sports Medicine and Science—Part I: Sports Medicine and Science Issues and Trends—Univ. of North Carolina, Chapel Hill
July 13-15	Olympic Festival Congress on Sports Medicine and Science—Part II: Athletic Injuries Prevention and Treatment—Duke Univ., Durham, N.C.
July 13-15	NIH Consensus Development Conference on Neurofibromatosis—Bethesda, Md.
July 21-26	World Association of Societies of Pathology (Anatomic and Clinical)—Sheraton Hotel, Washington, D.C.
July 29-Aug. 3	International Doctors in Alcoholics Anonymous—Lexington Marriott Resort, Lexington, Ky.

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World Blindness

ROGER L. HIATT, M.D.

We do not develop any profession in isolation, any more than we can live our religion in isolation. There are many problems facing us as physicians, just as there are many problems facing us in applying our religious faith to daily life. Coupled with disease is poverty and ignorance, and superimposed on top of that is political oppression in many areas of the world.

The changes that face us as ophthalmologists are similar to those facing other branches of medicine. One change, of course, includes the breakdown of the family and the increase in divorce. Another is the problem of drug use and abuse. Then, too, there are the problems of pornography and crime.

The problems of world health concern us all. In Nigeria, for example, the life expectancy is 41 years for men, but in our country it is 74 years. It has been shown that in some of the developing countries, 50% of the children never see their fifth birthday. In some of these countries there is one doctor for every 200,000 people, where in New York City there is one doctor for every 689 people.

If these problems were not enough, we have the problem facing our profession of regulation of hospitals and practices by government and other organizations. Then there are the internal stresses in our own fields. We have had stresses in ophthalmology, including the discussion of whether it is right to dispense glasses and the merits of phacoemulsification or intraocular lenses. In addition, advertising has become more commonplace. The problem of depression and suicide among ophthalmologists is reported to be second only to that among psychiatrists. Then there is the malpractice issue and the increase in the cost of malpractice insurance, coupled with the inroads by nonmedical practitioners into the practice of medicine. The subject that should be of most concern to us as ophthalmologists, and the heart of international problems, is world blindness. The water is not "getting to the end of the row."

In America there are about half a million blind people: to that number we add approximately 30,000 each year. We have about 2 million people in America with glaucoma, but half of them do not know they have it, and go untreated. Blindness is a major cause of disability in America today, where the common causes of blindness include cataract, glaucoma, diabetes, trauma, and retinal degeneration; some of these are very uncommon in foreign countries.

The prevention of avoidable blindness is a task so formidable and so urgent that the international community must constantly search for ways to do the job better and faster. Currently, logistical, political, financial, and cultural constraints

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From the Department of Ophthalmology, University of Tennessee College of Medicine, Memphis.

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inhibit the wholesale application of existing blindness—prevention technology that could save the sight of 80% of people destined to lose it.

Clearly, we must find preventive measures that can be made available to large populations more readily and more effectively than those in our present armamentarium. We must also find ways to overcome barriers to the delivery of care.

The Blinding Diseases

Four diseases are of such magnitude in developing countries that their control would reduce blindness close to the 0.2% prevalence rate now found in the United States; they are cataract, trachoma, xerophthalmia and onchocerciasis.

Cataract affects 17 million people; it clouds the lens and blocks vision.[1] Each year more than 1 million persons lose their sight this way. In India, where there are millions of people blind from cataract, there are only 3,500 ophthalmic surgeons[2]; in America we have 12,000 ophthalmic surgeons. In Bangladesh there are a million people blind from cataract, but they have only 25 ophthalmic surgeons.[2] In Pakistan a patient can be operated on for cataracts for $10, but some people do not have the $10.

Trachoma is a chronic, highly contagious infectious disease that afflicts 400 million people and has blinded 6 million in areas where poor sanitation allows the trachoma virus to thrive and spread.[1] Trachoma is the single most preventable cause of blindness in the world. Antibiotic ointment is an effective treatment. In Saudi Arabia, the country with all the oil, one can cure trachoma with 50 cents worth of antibiotics administered at the right stage, but many people do not have the 50 cents; because of customs or some other obstacles, others do not take the medication given them.

Each year *xerophthalmia* destroys the eyes of 500,000 malnourished children under the age of 6. Though megadoses of vitamin A at six-month intervals can prevent the disease,[1] there are a million children in the third world blind every year because they do not have enough vitamin A. Remember, if we don't get the "water to the end of the row," it is because the patients don't have the 90 cents worth of vitamin A and the protein rich diet to go with it to prevent the blindness.

Onchocerciasis, the "river blindness" of the southern half of the globe, comes from a parasite transmitted by black flies, which breed along fast flowing streams. Of 100 million infected people, 1 million have reached the blinding stage of the disease. The only control measure is spraying fly-breeding sites with insecticide.[1] There are 60 million people around the world with onchocerciasis; in some of the villages, half of the people have the disease.

Scope of the Problem

The World Health Organization (WHO) estimates the total blind population at 42 million, which is likely to double by the year 2000 unless urgent research programs are instituted to control the causes of visual disability.[3]

Two basic factors are at work. More than three-fourths of the blind live in developing countries, beyond easy reach of basic eye care services; 80% of blindness would be preventable or easily curable through appropriate epidemiological studies and development and application of new technologies for treatment and cost-effective delivery.

Developing countries have a disproportionate share of the world's 42 million blind people because poverty, poor sanitation, malnutrition, environmental hazards, infection, and absence of eye care inevitably lead to a high prevalence of blindness and serious visual impairment. In the third world, blindness rates are often 10 to 40 times higher than those of the industrialized West, where many of the blinding eye disorders have been eradicated as a result of better health care and improved standards of living.

The economic cost of blindness is staggering. WHO estimates the minimal annual cost in the third world to be $1.1 billion.[3] Families must assume the cost of feeding, clothing, and sheltering the blind person, as well as provide someone to guide him. The blind individual who is economically unproductive and whose needs nevertheless must be met represents an economic loss equal to the productivity of two or three persons.

A global program to reduce preventable blindness would add millions of people to the work force, thereby improving productivity, encouraging economic development, and easing the strain on beleaguered economies that are otherwise forced to commit scarce development resources to social services for millions of blind and visually handicapped people. While it is unrealistic to expect complete eradication of blindness, the recurrent cost of maintaining control programs probably would be reduced to $100 million to $200 million annually.[3]

Cataract

Cataract is the leading cause of blindness in the world, with 1.25 million new cases each year.[3] Cataract formation cannot be prevented by any known means, but sight can be restored by surgical removal of the opacified lens. Sixteen million of the 17 million people with cataract reside in the developing areas of the world; they are blind because of the lack of trained medical personnel and inadequate access to surgical services.

Life expectancy is on the rise in developing countries; the predicted fivefold increase in the number of persons over 55 years of age in these countries by the year 2025 means a significant growth in the magnitude of cataract blindness unless full-scale control programs become operational.[4]

Blinding Trachoma

Trachoma, one of the most common diseases in the world, is a major public health problem, affecting 400 million persons, most of whom live in poor rural communities of the developing world.[3] Approximately 6 million have been blinded by the disease; a far greater number suffer partial loss of vision.

Trachoma is a chronic, contagious, inflammatory ocular disease that leads to scarring of membranes covering the eye and the inside of the eyelids, to deformities of the eyelids, and to corneal opacity. The specific cause is a microbial pathogen, *Chlamydia trachomatis,* which is transmitted person to person by close contact and by flies, which are common in the region.

The highest prevalence rates of trachoma occur in parts of Africa, the Middle East, the dryer regions of Southeast Asia, and the Indian subcontinent. It thrives in the poorest villages, where sanitation and personal hygiene are very low. In hyperendemic communities, upward of 90% of the population has trachoma. In such heavily affected locales, almost all children are infected by the age of 2. They constitute the main reservoir of the disease.[5]

Present control programs of modest effectiveness include mass application of topical antibiotics and surgical intervention to correct eyelid deformities. Such control programs could, over a decade of effort, sharply reduce the prevalence of trachoma and resultant blindness. There is evidence to suggest that research on developing a vaccine for trachoma control should be considered.

Xerophthalmia and Measles-Associated Blindness

One of the oldest recorded human afflictions, xerophthalmia remains a major clinical and public health problem.[3] An easily treated disease, this nutritional disorder affects the most economically deprived populations of the developing world. Each year more than half a million children are blinded by this disease, which ranks among the five most common nutritional deficiencies.

Xerophthalmia and measles-associated blindness are the major causes of child blindness in the developing world. Xerophthalmia, also known as nutritional blindness, results from inadequate consumption of the vitamin A needed for healthy development of the eye, often in association with generalized chronic undernutrition. Protein deficiency interferes with storage and utilization of vitamin A. Anything that reduces intake, absorption, storage, or transport of vitamin A, or such a disease as measles that increases the metabolic needs for vitamin A, will contribute to or precipitate xerophthalmia. At least 5 million children in Asia alone suffer clinically detectable xerophthalmia every year, and approximately 50% of these children develop potentially blinding corneal destruction; 250,000 are blinded in both eyes. The total number of children with xerophthalmia throughout the world is upward of 10 million.

The immediate treatment for xerophthalmia is rapid replenishment of vitamin A by the oral administration of high potency (200,000 IU) vitamin capsules and proper attention to protein-calorie malnutrition, as well as to any underlying systemic infection.

The most widely practiced short-term intervention measure in endemic regions is periodic oral administration of high-dose vitamin A to the most vulnerable population groups.

Long-term prevention and proposed control programs involve broad changes in infant and child care and feeding practices leading to increased consumption of vitamin A-rich foods such as green leafy vegetables and yellow fruits.

Measles is a precipitating cause of xerophthalmia in some Asian and African children; measles per se, however, is considered the leading cause of child blindness in Africa. While it is thought to be linked to malnutrition, the etiology of measles-associated blindness is still unproven, and the possibilities of effective control are currently limited to measles immunization.

Onchocerciasis

Onchocerciasis, a blinding parasitic disease, now infects upward of 50 million people[3]; 1 million are totally blind as a result. A filarial parasite is spread from person to person by repeated bites of the black fly. The individuals at greatest risk and most in need of treatment are those who live or work closest to breeding sites; they are typically male, aged 15 to 45 years. In some communities it is the women whose activities bring them into contact with the breeding sites, and who, therefore, have greatest exposure to the disease-carrying flies.

This disease, commonly known as "river blindness," occurs near fast moving rivers and streams in equatorial Africa, and parts of Mexico and Central and South America. In some African villages almost half the adult population has been so blinded.[3]

In villages where onchocerciasis is widespread, no less than 40% to 50% of the adult population are blind, handicapping not only the affected individuals, but the community's agricultural productivity as well.

The human body is the only place in which the adult worms live. The adult female releases millions of microfilariae that invade tissues and organs, including the skin and eyes, creating pathological tissue changes. The onchocercal eye lesions are the most serious of the resulting lesions because they cause visual impairment and blindness. The only known effective preventive measure is avoidance of the black fly's breeding sites. The current strategy for prevention, therefore, is to control the black fly population by the application of insecticides for a period of approximately 20 years. At the end of this time, the vector should be almost eradicated from the region.

Treatment of an individual afflicted with onchocerciasis is aimed at preventing worsening of the disease by eliminating the adult worms and microfilariae from the infected person without serious adverse reactions. This is not easy because currently available drugs are too toxic to be used on a large scale. If it were possible to identify those at risk of blinding infections at an early stage, a nontoxic filaricide could provide effective treatment.

Summary

There are things we can do individually and collectively to erase such tragedies from the world in which we live. It was Lincoln who said "a nation cannot permanently endure half slave and half free." I would like to paraphrase that and say that a world cannot exist half well and half sick, or half blind and half sighted. Many of our international problems are traceable to disease, poverty, and ignorance, and not just to the wars that come from political oppression. We cannot do everything, but what we can do we ought to do. It is a pity to live in a world that is blind. It is a pity to live in darkness where so much light and truth is available. "Where there is no vision the people perish." Blindness comes from a lack of freedom also—freedom to live every day and feel good, and freedom from fear, want, and hunger.

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Blindness From Diabetes in Tennessee

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For more than a decade, diabetes mellitus has been described as a major cause of visual impairment in the United States.[1] It is estimated that more than 5,000 new individuals in the United States become blind from diabetes each year.[2] In Tennessee, when all causes of blindness are combined, we expect fewer than 1,000 new blind each year[3]; how many of these are blinded by diabetes is not known. The following study was initiated to determine the influence of diabetes mellitus on the Tennessee blind population.

Exact data on blindness is difficult to ascertain in Tennessee because some patients with visual impairment are outside of the mainstream of medical care. On the other hand, many blind persons in Tennessee have their name, diagnoses, and other demographic data on file with the Registry for the Blind. This record is maintained by the Tennessee Department of Human Services' Division of Services for the Blind. The Registry facilitates the Division in its programs of assistance from local, state, and national sources. In an attempt to determine the impact of diabetes mellitus on blindness in Tennessee, we analyzed the data in the 1986 Registry.

Method

In July 1986, the Registry for the Blind contained data on 7,188 persons, each of them registered as blind when the best corrected visual acuity in the better seeing eye was 20/200 or worse, and/or the visual field was restricted to 20 degrees or less. A systematic representative sample was obtained by reviewing every sixth file, and determining for each individual the date of birth, age of onset of blindness, causes of blindness, and geographic location of each patient.

Results

The records of 1,198 blind persons were reviewed in detail. A single cause of blindness could not be determined in all cases, in many patients because there were multiple disorders in the involved eyes, any one of which could have been the major contributor. Though in many patient records, the data did not separate primary from secondary features, in those individuals in which a single, or primary, cause could be ascertained, the blindness was caused by the processes listed in Table 1.

The age distribution of blind diabetics demonstrated a peak in seventh decade; there were no individuals under 30 years of age blind from diabetes in the sample population (Table 2). Di-

TABLE 1

CAUSES OF BLINDNESS IN 1986

Cause	%
Cataract	19.6
Glaucoma	13.5
Optic atrophy	12.1
Diabetes mellitus	9.7
Trauma	7.5
Retinitis pigmentosa	6.8
Macular degeneration	5.2
Nystagmus	4.3
Retinal degeneration	3.9
Myopic degeneration	3.8
Retinopathy of prematurity	2.9
Chorioretinitis	2.1
Optic neuritis	1.8
Retinal detachment	1.4
Histoplasmosis	1.3
Albinism	1.3
Amblyopia	1.2
Neoplasms	0.9
Toxoplasmosis	0.7

TABLE 2

AGE OF BLIND DIABETICS IN REGISTRY

Years of Age	% of Total
80 to 90	14.8
70 to 80	16.7
60 to 70	38.9
50 to 60	9.3
40 to 50	9.2
30 to 40	7.4
Unreported	3.7

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TABLE 3

AGE OF ONSET OF BLINDNESS IN DIABETIC PATIENTS IN 1986

Years of Age	% of Total
Under 25	9.4
26 to 35	12.5
36 to 45	15.6
46 to 55	34.4
56 to 65	28.1

TABLE 4

DISTRIBUTION OF BLIND DIABETICS BY REGION

Region	% of Blind Diabetics
Memphis	22
Jackson	13
Chattanooga	13
Paris	11
Nashville	11
Johnson City	9
Knoxville	9
Columbia	7
Cookeville	4
Unspecified	1

abetic blindness appears more commonly in women than in men, with a ratio of 4 to 1.

Information regarding the duration of visual impairment was not available in every record, but in those records that had these data listed, blindness due to diabetes occurred most often in patients in whom the onset of blindness was between the ages of 46 and 54. Less than 10% of diabetics in the Registry began losing their vision before 25 years of age (Table 3), though more than one-third of the records had no data regarding age of onset.

The geographic distribution was examined using the first three digits of the zip code of each patient. In this manner, Tennessee can be divided into nine regions, identified by a well-known community in each. The distribution of blind diabetics was greatest in the Memphis region and lowest in the Cookeville region, but trends relating to urban-rural differences and trends reflecting physician distribution could not be detected from the data (Table 4).

Discussion

Since 1978, when the first evidence that laser surgery can reduce blindness from diabetes was published,[4] there have been numerous studies to determine its impact,[3,5,6] but many years of diabetes mellitus are needed before potentially blinding changes begin. Many patients therefore appear to drop out of the mainstream of treatment before the time of greatest visual danger.

In Tennessee, though the ratio of women to men in the Registry for the Blind is equal (female 49.8%, male 50.2%), in diabetic blindness in Tennessee, this ratio is surprisingly 4 to 1 (female 79.6%, male 20.4%). In other states, the female to male ratio of diabetics with eye disorders has been nearly equal.[3,6,7] Why Tennessee has this uncommon ratio could not be explained from the available data.

In Tennessee, most diabetic blindness began between 60 and 70 years of age; this was not much different from ages reported in other states.[3,6,7] In other studies, blindness was found in about 10% of patients after 20 years of diabetes mellitus; this percentage increases by about 10% more for each decade among survivors,[6,7] which may account for the peak in this study after age 60. Though others have indicated a difference in blindness rates between juvenile onset and adult onset diabetics,[6,7] our data were unable to confirm their reports. That only 37% of the patients in Tennessee began to lose their vision before age 45 may be due to such things as the short life expectancy of juvenile diabetics, rather than the visual disorder.

Although the largest percentage of blind diabetics lived in the Memphis region, other urban regions were not much different from corresponding rural regions. For this reason, we believe the Memphis peak is related to the larger population base there. We could not demonstrate a relationship between the availability of medical care and the incidence of diabetic blindness in our state.

Acknowledgment

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Intussusception in Pregnancy

A Case Report

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Introduction

Intussusception is an extremely rare cause of intestinal obstruction in adults. According to some authors,[1] it accounts for only 5% of mechanical intestinal obstruction, whereas in children, intussusception accounts for approximately 80% to 90% of obstruction.[2]

Bourque and Gibbons,[3] reviewing the literature from 1914 through 1975, found only 13 cases of intussusception complicating pregnancy. They reported two additional cases from their institution.

We present a case of intussusception in pregnancy at 12 weeks' gestation, review the literature, and discuss problems in diagnosis and management.

Case Report

A 21-year-old white woman, gravida 2, para 1, abortus 0, whose last normal menstrual period was July 6, 1982, presented herself at 6 weeks' gestation for prenatal care, having been previously delivered in 1980 at our facility. Her only initial complaint was nausea, for which Bendectin tablets were prescribed.

She returned to the office on Sept. 15, 1982, at 10 weeks' gestation complaining of acute onset of right lower quadrant pain associated with nausea, vomiting, and diarrhea. She was admitted for evaluation and subsequently underwent diagnostic laparoscopy to rule out ectopic pregnancy. Findings at laparoscopy were a normal appearing uterus of 10 weeks' size and a ruptured, bleeding right ovarian cyst. The cyst edges were cauterized with bipolar forceps, and the patient subsequently improved and was discharged on Sept. 17, 1982, much improved.

She returned to the office on Sept. 20, 1982, because her nausea and vomiting had not responded to Bendectin or Phenergan suppositories. She was admitted for treatment of hyperemesis gravidarum.

Examination revealed a thin white woman in mild distress and weighing 104 lb; temperature was 99.4°F, pulse 96/min, respirations 14/min, and blood pressure 190/70 mm Hg. Her general physical examination was within normal limits. Pelvic examination was compatible with a 10- to 12-week intrauterine gestation.

Laboratory assessment revealed a hematocrit of 41.5%, white blood cell (WBC) count 10,100/cu mm, with 82% segmented neutrophils, 15% lymphocytes, and 1% monocytes. Urine specific gravity was 1.008, pH 7, with negative ketones, 1 to 3 WBCs, and 1+ rods and cocci. A two-hour urine amylase was 54 U/dl (normal 4-37), LDH 164 U/L (normal 100-190), SGPT 77 U/L (normal 3-36), SGOT 51 U/L (normal 22-37), alkaline phosphatase 11.9 U/L (normal 2.5-9.7), bilirubin 0.4 mg/dl, and gamma-GT 88 U/L (normal 5-55). The blood amylase was 52 U/dl, BUN 5 mg/dl, sodium 131 mEq/L, potassium 2.9 mEq/L, chloride 86 mEq/L, and CO_2 32 mEq/L.

Ultrasonogram of the gallbladder was normal and pelvic ultrasound demonstrated intrauterine pregnancy compatible with 12 weeks' gestation.

The patient was given intravenous D10W and potassium, with Phenergan intramuscularly as needed for nausea. By Sept. 25, 1982, her abdomen had become distended and tympanitic, with diminished bowel sounds. A gastroenterology consultant thought that the most likely diagnosis was pancreatitis with ileus. Nasogastric suction was instituted and by Sept. 29, 1982, the patient was much improved. She had continued to pass flatus and have daily bowel movements.

On Sept. 28, 1982, nasogastric suction was discontinued and liquid feedings were resumed. Her diet was advanced, but on Sept. 30, 1982, colicky abdominal pain returned, with hypoactive bowel sounds and rushes. Esophagogastroduodenoscopy revealed reflux esophagitis, but was otherwise normal.

On Oct. 2, 1982, the patient began to have fecal appearing vomitus, and supine and upright abdominal films were compatible with small bowel obstruction. On Oct. 2, 1982, an exploratory laparotomy was performed, and when an ileocecal intussusception could not be reduced, a resection and end-to-end anastomosis was performed. Postoperative recovery was very rapid and she was discharged on the seventh postoperative day.

The surgical specimen showed benign focal polypoid lymphoid hyperplasia of the terminal ileum causing intussusception of the ileum into the cecum and ascending colon, resulting in acute necrotizing enteritis. There was hemorrhagic necrosis of the terminal ileum, and acute visceral peritonitis of the terminal ileum, cecum, and ascending colon, with periappendicitis.

The remainder of the patient's antepartum course was uncomplicated, and she delivered a 7 lb 7 oz female infant at term on April 6, 1983. The postpartum course was unremarkable.

Discussion

The diagnosis of intestinal obstruction from any cause in early pregnancy is made more difficult by the common complaint of nausea and vomiting, which may persist until the second trimester. A second difficulty in diagnosis is the reluctance to expose an early pregnancy to ionizing radiation; these together contributed great-

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ly to a delay in diagnosis in our patient and in the two cases reported by Bourque and Gibbons.[3] In the nonpregnant patient, nausea and vomiting of any significance is initially evaluated with abdominal x-rays as a matter of course, thereby facilitating diagnosis. In our patient, the diagnosis was not made until gastrointestinal rushes and fecal vomitus pressed roentgenographic assessment.

According to Bourque and Gibbons,[3] adhesive bands account for some 60% of intestinal obstruction occurring during pregnancy. Volvulus is next most common and intussusception third.

The etiology of intussusception in children is generally idiopathic (Todani, Tabushi, and Tanaka[4] have even reported one in-utero intussusception). Intussusception in the adult is usually associated with gastrointestinal lesions, among them Meckel's diverticulum and benign or malignant tumors. Aston and Machleder[2] reported a case of intussusception in a 15-year-old girl at 12 weeks' gestation with a cavernous hemangioma of the distal ileum at the intussusceptum.

Although the postoperative course of pregnant patients operated upon for intussusception may be uncomplicated, as in our patient and the patient of Aston,[2] the two cases reported by Bourque and Gibbons[3] demonstrate that difficulties in diagnosis may lead to disastrous results. Their first patient required two exploratory laparotomies and ultimately delivered a stillborn premature infant, and their second patient underwent cesarean section and a subsequent laparotomy was required for diagnosis. We suspect that in our patient the pain that prompted the diagnostic laparoscopy was most likely caused by intussusception, and not the ovarian cyst.

Summary and Conclusions

We present this case as an addition to the small series of cases of intussusception in pregnancy as a reminder that all nausea and vomiting in early pregnancy is not necessarily hyperemesis gravidarum. A high index of suspicion and judicious use of timely abdominal x-rays may provide early diagnosis and avoid a potential catastrophe.

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Acute Interstitial Nephritis Induced by Cimetidine

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Introduction

The therapeutic efficacy of cimetidine (Tagamet) in peptic ulcer disease has been well established.[1-3] With its widespread use, various adverse effects have been increasingly recognized.[3] Acute interstitial nephritis with progressive renal failure[4-11] is one of the most important adverse effects for clinicians to be aware of. We report a case of biopsy-proven, cimetidine-induced acute interstitial nephritis, review eight other cases of cimetidine-induced acute interstitial nephritis, and discuss the clinical features and immunological mechanisms.

Case Report

A 48-year-old white man was seen by a physician on March 8, 1984 because of heartburn. His urine at that time did not reveal proteinuria or hematuria. Cimetidine 300 mg orally four times a day was prescribed. On April 4, 1984, he developed nausea, vomiting, cough, and headache. White blood cell (WBC) count was 10,650/cu mm. Hematocrit was 36%. Urinalysis revealed 2+ protein, 10 to 14 WBCs per high power field and 3 to 5 RBCs per high power field. He was treated with amoxicillin 250 mg three times a day, but despite the treatment, he became more ill with chills, temperature of 101°F, suprapubic pain, and bilateral flank pain. He was admitted on April 9, 1984, to Bradley County Memorial Hospital for further evaluation.

Physical examination upon admission revealed a well-developed young white man in acute distress with chills. Blood pressure was 150/90 mm Hg, pulse 104/min, temperature 102.5°F, respiration rate 24/min. The positive physical findings were limited to suprapubic and bilateral flank tenderness.

Laboratory tests showed hemoglobin 10.8 gm/dl, hematocrit 33%, WBC count 11,600/cu mm, with 88% neutrophils, 6% lymphocytes, 5% eosinophils and 1% band forms; serum calcium was 8.3 mg/dl, inorganic phosphorus 3.3 mg/dl, glucose 108 mg/dl, BUN 17 mg/dl, creatinine 1.9 mg/dl, uric acid 5.8 mg/dl, cholesterol 179 mg/dl, total protein 7.1 gm/dl, albumin 3.8 gm/dl, bilirubin 0.8 mg/dl, alkaline phosphatase 329 U/L, LDH 142 U/L, SGOT 55 U/L, GGT 192 U/L, serum sodium 136 mEq/L, potassium 4.9 mEq/L, chloride 104 mEq/L, CO_2 18 mEq/L. Urinalysis showed 5 to 10 RBCs and 75 to 100 WBCs per high power field. Reticulocyte count 0.3%, erythrocyte sedimentation rate 130 mm/hr, serum iron 2 μg/dl, total iron binding capacity 241 μg/dl, and serum folate and serum B_{12} were normal. Multiple urine and blood cultures grew no organism, and intravenous pyelogram was normal. Chest x-ray was normal, cold agglutinin was negative, and malaria smear was negative. Occult blood in stool was 3+, but there were no ova or parasites, and stool culture was negative. Upper gastrointestinal x-ray revealed large hiatal hernia with reflux.

With a presumptive diagnosis of acute pyelonephritis, ampicillin and tobramycin were initiated intravenously. Cimetidine 300 mg was continuously given every six hours orally. Despite the antibiotic treatment the patient had a persistent, spiking temperature up to 102°F daily. Serum creatinine reached to 5.7 mg/dl on April 16, 1984. Multiple complete blood counts did not reveal eosinophilia. No rash was observed. On April 15, 1984 cimetidine was discontinued and ranitidine was initiated with a dose of 150 mg twice a day. Serum creatinine gradually declined to 2.8 mg/dl on April 21, 1986. After discharge from the hospital he took cimetidine for three or four days, and again developed fever, back pain, pyuria, hematuria, and proteinuria. His BUN and creatinine went up to 40 and 3.9 mg/dl, respectively, and he was found to have eosinophilia (720/cu mm). The clinical course of this patient is summarized in Fig. 1.

On May 14, 1986, he was referred to a nephrologist (T.T.O.), who suspected acute interstitial nephritis due to cimetidine. The patient did not take cimetidine for a week prior to this visit. Percutaneous renal biopsy was performed on May 19, 1984. Light microscopic examination revealed normal glomeruli except for increased mesangial matrix. There was

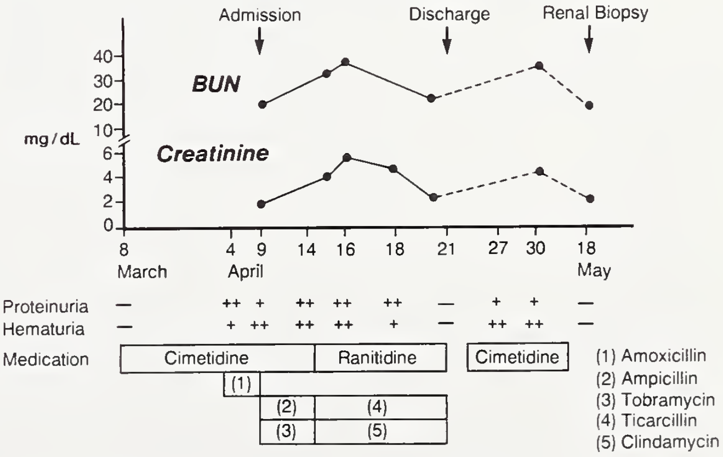

Figure 1. Summary of clinical course of patient with cimetidine-induced acute interstitial nephritis.

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a diffuse interstitial infiltrate with predominantly plasma cells and a few lymphocytes, eosinophils and neutrophils, and a diffuse interstitial fibrosis. Immunofluorescent microscopic examination revealed no deposition of immunoglobulin G, M and A, C1q, C3 or fibrin reactive products. Electron microscopic examination revealed increased mesangial matrix and interstitial collagen and tubular degeneration with numerous plasma cells and lymphocytes. No election dense deposits were observed. At the time of renal biopsy, the urine did not reveal pyuria, hematuria, or proteinuria. BUN and creatinine were 17 and 2.6 mg/dl, respectively. A short course of prednisone therapy was prescribed, but he moved to another state, and no further follow-up was made.

Discussion

It is known that a mild increase in serum creatinine commonly occurs in patients taking cimetidine,[1-3,12] but the values rarely exceed 2 mg/dl and promptly return to baseline values when cimetidine is discontinued.[12] Although the mechanism by which cimetidine increases serum creatinine is not known, it is suggested that cimetidine competes with creatinine in its excretion through the kidney.[12]

Acute interstitial nephritis has been recognized with cimetidine therapy. The incidence was estimated at less than 0.1/100,000 patients on the basis of worldwide spontaneous reporting system.[13] As with most spontaneously reported adverse reactions, this is probably an underestimation, but the magnitude remains to be determined. The clinical manifestations in most patients with documented interstitial nephritis include fever, chills, malaise, night sweat, myalgia, mild eosinophilia, sterile pyuria, proteinuria, hematuria, and evidence of decreased renal function after a few to several weeks of cimetidine therapy.[4-11] Interestingly, rash has not been observed in these patients. Improvement promptly followed discontinuation of the drug.[4-11] Some patients were treated with steroids, but the role of steroids in these patients remains to be defined. The clinical features of biopsy-proven cimetidine-induced acute interstitial nephritis are summarized in Table 1. Our patient presented chills, fever, leukocytosis with mild eosinophilia, sterile pyuria, hematuria, proteinuria, and progressive azotemia. The laboratory tests did not disclose any infectious etiology. The patient made a significant improvement after discontinuation of cimetidine therapy. Inadvertently, this patient was rechallenged with cimetidine, resulting in recurrence. Renal biopsy revealed acute and chronic interstitial nephritis. Cimetidine was the single drug that this patient was taking when his renal disease developed. These observations strongly indicate cimetidine as the cause of acute interstitial nephritis in this patient.

Cimetidine has been reported to cause immediate-type hypersensitivity reactions with urticaria[14] despite its use in treating urticaria. The absence of rash in any of these patients with acute interstitial nephritis excludes a role for immediate-type hypersensitivity reaction in the patho-

TABLE 1

CLINICAL FEATURES OF CIMETIDINE-INDUCED ACUTE INTERSTITIAL NEPHRITIS

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Our Case
Length of therapy	2 wks	3 wks	4 wks	4 wks	3 mos	2 mos	5 mos	2 wks	4 wks
Fever	+	−	+	−	+	+	+	−	+
Myalgia	+	−	+	−	−	−	+	−	−
Proteinuria	+	−	?	+	−	?	2+	+	2+
Hematuria	+	−	?	−	?	?	−	−	2+
Pyuria	+	−	+	+	?	?	+	+	+
Leukocytosis	+	+	−	−	+	−	−	+	+
Eosinophilia	+	+	+	−	+	−	+	+	+
BUN (highest)	37	81	46	68	?	244	198	40	32
Creatinine (highest)	2.8	7.9	4.6	8.1	5.6	16.4	19.6	4.1	5.7
Steroid therapy	No	Yes	No	No	No	No	Yes	Yes	Yes
Recovery	C	P	P	P	C	P	C	P	P
Associated condition	DU	RE	HH	EE	E	DU	DU	UE	HH

KEY: + positive; − negative; ? unknown; C complete; P partial; DU duodenal ulcer; RE regional enteritis; HH hiatal hernia; EE erosive esophagitis; E esophagitis; UE ulcerative esophagitis.

genesis. The absence of immunoglobulin deposition and the presence of predominant round cell infiltration in animal models of acute interstitial nephritis mediated by delayed hypersensitivity[15] are similar to the observations in our patient and others. In-vitro and in-vivo studies have clearly demonstrated that cimetidine is a potent stimulator of delayed hypersensitivity,[16] suggesting that our patient's renal disease may be mediated by cimetidine-induced delayed hypersensitivity.

From our experience and that of others we would recommend that a urinalysis and blood urea nitrogen or serum creatinine be done periodically in patients being treated with cimetidine, since early recognition of this renal disease and discontinuation of cimetidine provides complete resolution of the disease.

Acknowledgment:

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Special Item

Trauma Systems Development:
A Proposal for the State of Tennessee

JAMES W. PATE, M.D.; TIMOTHY C. FABIAN, M.D.; and
C. RICHARD PATTERSON, M.D.

Tennessee and the rest of the nation have been brought to a critical point in community responsibility and decision-making regarding the care of seriously injured trauma victims. Several factors are responsible for our arrival at this point, and these are the focus of this article. The purpose of this report is to review these factors and present a concept which would provide every trauma victim in Tennessee access to an appropriate level of care within one to two hours of injury.

The first factor influencing our current situation is the widening realization of the terrible societal cost exacted by trauma, which is the leading cause of death among Tennesseans and other Americans under the age of 40.[1] The cost of the loss of these citizens and their potential contribution to our communities is virtually incalculable, and the cost in dollars expended for trauma care, including that of resulting disability, exceeds the cost of care for heart disease or cancer, or any other disease. Although the realization of the impact of trauma is widening, it is not yet mature in the public mind. By way of illustration, the Tylenol poisonings caused seven deaths in one week and resulted in massive publicity, concern, product withdrawals, and a concerted effort through expensive product redesign and repackaging to prevent recurrence. During that same week, 900 Americans were killed in automobile accidents and approximately 2,000 were permanently disabled. The trauma toll continues week after week, without proportionate attention and effort.

A second factor is the growing sophistication in the expertise pertinent to the care of trauma victims. The body of knowledge, both basic and clinical, regarding the prevention and management of shock, sepsis, multiple organ injury and failure, and skeletal and neurologic injury has expanded tremendously in the past few years. This level of expertise is *essential* to proper care for the 5% to 10% of trauma victims who are the most seriously injured and who account for the majority of deaths. Rather than being relegated to the occasional practice of routinely trained physicians, the care of such major trauma patients is increasingly being recognized as a practice for teams of surgical and nonsurgical specialists.

Although trauma surgery has become more sophisticated and complex, the majority of general surgeons finishing their residencies are poorly trained in trauma care. A review of 933 applicants to the American Board of Surgery in 1980 demonstrated that 18% had managed fewer than 10 trauma victims, 47% fewer than 20, and 67% fewer than 30.[2] Trauma was found to constitute *less than* 5% of the operative experience for 95% of the American Board of Surgery applicants. Furthermore, the American College of Surgeons recommends that a properly trained surgeon manage a minimum of 50 trauma cases a year in order to maintain the skill necessary to the practice of traumatology. The number of surgeons with this continuing breadth of experience is very limited, and major trauma care is really incompatible with the usual elective surgical practice. Victims of multiple systems trauma can receive optimal care only at regional centers where the valuable and limited resource of expertise in trauma can be focused.

The burgeoning knowledge in trauma care has led to the third factor influencing our current societal dilemma: The realization that many trauma deaths and disabilities are preventable. It became apparent during the Vietnam War that,

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From the Department of Surgery, University of Tennessee, Memphis.

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given comparable levels of injury, a United States citizen had a better chance of survival in a jungle 5,000 miles from home than on an American highway. Cales and Trunkey[3] recently reviewed the published studies of trauma deaths in the United States. Among the 4,720 trauma victims in 26 reports from localities without organized trauma care, the preventable death rate was 23%. When the principles of rapid transportation, well-planned triage, and dedicated and experienced trauma care facilities are translated from the wartime experience into civilian circumstances, substantial reductions in mortality rates can be accomplished. In Orange County, California, for example, the preventable death rate before the institution of a coordinated system of trauma care was 54%. After the medical and political leaders of that community established a system of designated trauma facilities and triage protocols, the preventable death rate dropped to 4%.[4] Similar reductions in preventable deaths have been experienced in other, disparate communities.

A fourth important factor is that the elements of a successful system of trauma care have been defined and are readily available to those wise enough to implement them. The American College of Surgeons, after years of study by experts empaneled from across the nation, established the guidelines that enable any system to offer the best available trauma care.[5] The state of Tennessee, through the Licensing Board for Health Care Facilities, has adopted but not yet implemented a designation process that follows the guidelines of the American College of Surgeons. That process describes the *minimal* standard that should be achieved and maintained before an institution is designated a "trauma center." A Level I Trauma Center, the most sophisticated level of designation for example, must be capable of managing any type of trauma at any hour, every day. Surgical teams, including trauma, neurosurgery, and anesthesia, must be present in-house, and other surgical specialists (orthopaedic, cardiovascular, plastic and reconstructive, otolaryngology, and ophthalmology) must be available within minutes on a rigid call schedule. There must be continuing research programs in all areas, and the Level I Trauma Center must maintain active teaching programs for surgeons, nurses, and paramedical personnel in all specialties.

The Level I physical facility must be sophisticated and provide dedicated and *immediately* available operating rooms, critical care beds, x-ray facilities, including CT and angiography, and the personnel and equipment necessary to their rapid and efficient utilization. A Level I Trauma Center must also maintain a critical number of admissions (600 to 1,000/year) in order to maintain and improve its proficiency in the management of the seriously injured. The state of Tennessee has the necessary information available for the creation of a credible trauma system and already has in place the rudiments.

The state of Tennessee also enjoys a geography well suited to a trauma system, which is the fifth factor that has brought us to this point of opportunity. The two critical elements of a large population base and an academic infrastructure required for the support of a Level I Trauma Center are well-distributed in our state

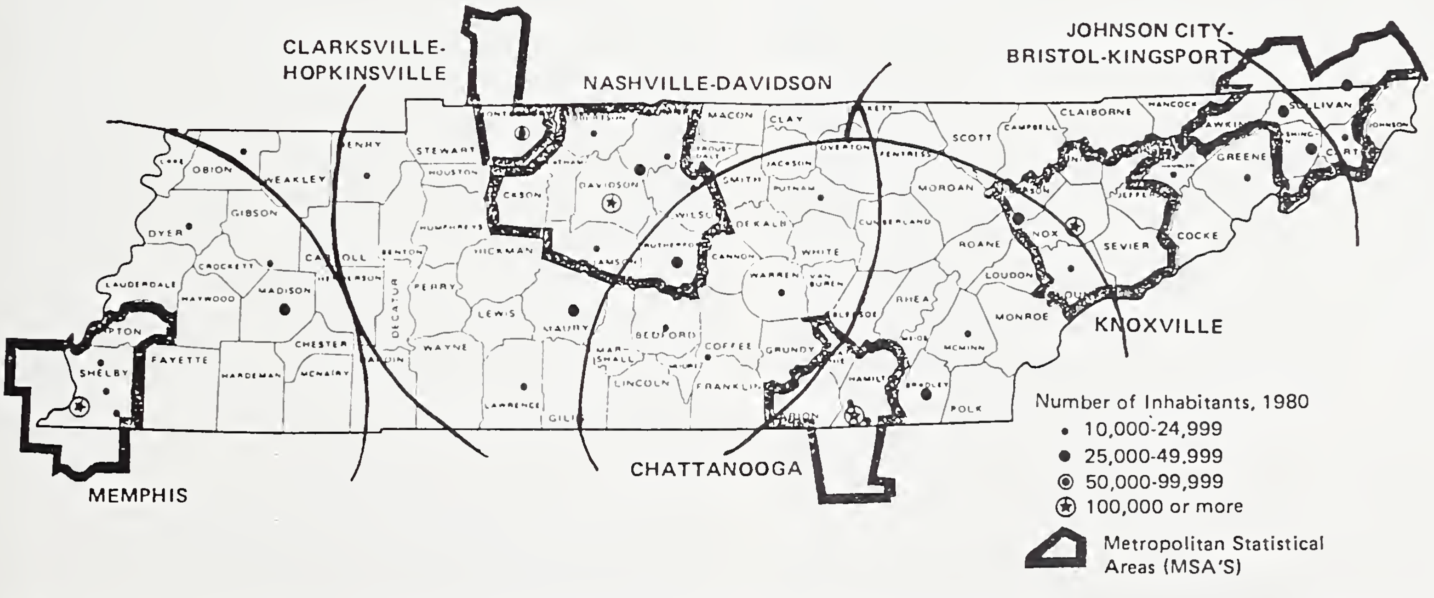

Figure 1. Map of Tennessee demonstrating a hypothetical trauma system with Level I Trauma Centers based in population centers and academic health centers. Arcs represent areas about each center from which patients could be reasonably transported to the trauma center in approximately one hour from injury. Level II centers, as in Jackson, could easily cover areas not convenient to Level I centers.

(Fig. 1). Based on authoritative projections of 1,000 serious injuries per 1 million population per year, Tennessee's population of 4.7 million would justify the construction of four or five Level I Trauma Centers. Service could also be provided to citizens of contiguous states by those centers. Medical schools and their affiliated clinical education centers are present in our major population centers: Memphis, Nashville, Chattanooga, Knoxville, and the Tri-Cities; these communities currently serve as regional referral centers for Tennessee and our neighbor states.

Level II Trauma Centers, appropriate for the care of the less severely injured, can be supported in cities such as Jackson, with a smaller population base and without major research and residency programs. Level II centers differ from Level I centers in that they require immediate on-call rather than in-house surgical, neurosurgical, and anesthesiology coverage. It should be emphasized that 90% of trauma victims can be cared for outside of Level I Trauma Centers, and overbuilding such facilities results only in harm. By matching regional resources to regional needs, the entire state could be linked by communication and transportation systems based at the Level I centers, with the result that every Tennessean would be within one hour of an institution appropriate for his level of injury, whether community hospital or trauma center.

A sixth factor, this time a negative one, is that trauma care is being appropriated by some as the latest means to increase "market share" in a medical atmosphere that is increasingly, and regrettably, commercial. Evidence of this trend is readily available across the country, where, in the absence of organization, competing hospitals engage in what can only be labeled "helicopter wars." This practice is unseemly at best, but in fact is worse, because it squanders resources without benefit to the patients or community it purports to serve. We must remember that a helicopter or any other single medical marvel is *not* synonymous with sophistication in trauma care. In order to save lives and prevent disability, resources must be arrayed in a coordinated system of trauma care, which includes strict evaluation, designation, communication, transportation, responsibility, and accountability through trauma registries.

Tennessee is therefore at a crossroads, a moment of great opportunity. We have a demonstrated need for improved trauma care, and we have the ability to provide that care. We must be alert to the danger of profligacy, however, for our ability rests primarily upon careful stewardship and coordination of our resources, and our opportunity may evaporate in the heat of undirected and self-serving competition. With a proper understanding and defense of the principles involved, Tennessee could serve as an example to the nation, as we have done in child safety-seat legislation.

An opportunity of this magnitude carries with it heavy responsibility, and the medical and political leaders of this state should cooperate to execute that responsibility. An example of such cooperation already exists in Shelby County, where the citizens have constructed a state-of-the-art facility with admitting and resuscitation areas, four operating rooms, recovery and critical care beds, and laboratory and x-ray facilities, all adjacent to each other and dedicated to the care of trauma victims. The local EMS Council was intimately and invaluably involved in establishing the transportation, communication, and triage protocols upon which this regional trauma system was developed. A single, multihospital-supported, but independent helicopter system serves the entire region, and multiple hospitals with triage and transfer of trauma patients directed from the communication unit in the trauma center.

The regional trauma center is staffed by the faculty of the University of Tennessee, Memphis, and is supported by their residency and research programs. Educational programs are continuously produced for physicians, nurses and other health professionals, and the emphasis in each profession is on continued progress and expansion of ability. This facility is capable of managing 3,000 to 3,500 seriously injured patients a year, and should be adequate for a population base of approximately 3 million. Facilities of at least half this size and capacity should be adequate for Level I centers in Tennessee's other major population centers. Five trauma centers throughout the state could handle massive trauma from a 9- to 16-million population base, and would serve as a resource for enriching trauma care through education in all other hospitals of the state, which would handle most injured patients.

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Unusual Presentation of Traumatic Transection of Aorta

GRIFFIN R. COATES, M.D. and DENNIS STAPLETON, M.D.

Case Report

A 66-year-old unrestrained white woman was involved in a high speed motor vehicle accident. Thirty minutes were required to extricate her from her automobile; her vital signs at the scene were blood pressure 170/80 mm Hg, pulse 100/min, respirations 24/min. She complained of left chest wall pain and left leg pain. After initial fluid resuscitation in the emergency department, palpable bilateral rib fractures were noted. There was no obvious sternal contusion, breath sounds were equal, and there was no cardiac murmur. Abdominal examination was unremarkable with no localized ternderness or rebound. Although she complained of pain and paresthesis in her left leg, there was no palpable pelvic or femur fracture, but there was decreased sensation on the left leg; the left femoral pulse was decreased and the left dorsalis pedis and posterior tibial pulses were not palpable. The skin below the left knee was mottled slightly. Cervical spine x-rays were normal. Chest x-ray demonstrated a widened mediastinum and the left third through seventh ribs and the right fourth and fifth ribs were fractured. There was a small left hemothorax. The pelvis, left femur, and left lower leg were normal on x-ray. The patient's hematocrit was 42% and hemoglobin 14 gm/dl; the urinalysis revealed 50 to 100 red blood cells per high power field.

A CT scan of the thorax and abdomen confirmed the widened mediastinum and did not show intra-abdominal solid organ injuries. After cardiothoracic consultation, a left tube thoracostomy was performed and the patient was transferred to Erlanger Hospital for arteriography. An anteriogram was initially attempted by a right femoral approach, but the radiologist was unable to pass the catheter retrograde up the abdominal aorta beyond the celiac trunk. The limited arteriogram revealed decreased flow to the left kidney, with very poor runoff and slow filling of the distal aorta. Using a right brachial artery approach, an abrupt cutoff of dye was found just below the level of the diaphragm, with decreased flow to the right kidney (Fig. 1). Initially the arch of the aorta was thought by the radiologist to be intact, but closer review revealed a linear defect as indicated by the arrow in Fig. 2, and in fact one could note delayed flow through the aortic arch despite adequate systemic blood pressure.

The patient was taken to the operating room where a left thoracotomy performed through the fourth intercostal space disclosed a large hematoma of the mediastinum. There was a pulse in the aorta to the level of the diaphragm. The aortic arch was examined proximal to the left subclavian artery, but since there was a large amount of hematoma in this area, before proceeding with further dissection the left femoral artery was exposed in preparation for left atrial to femoral artery bypass. Because of technical difficulties in cannulating the small fragile left atrium, through a median sternotomy the proximal ascending aorta was cannulated and connected with the left femoral artery through a Gott shunt. After the aorta was clamped just beyond the left carotid artery proximally and at the level of the diaphragm distally, the aortic hematoma and adventitia were opened, disclosing a circumferential tear at the aortic isthmus. All layers of the aortic wall could be found proximally, but just distal to the transection only the adventitia was present. When it was opened longitudinally, numerous intercostal arteries were noted to be sheared, and when the adventitia of the entire length of the descending thoracic aorta was opened distally there was still no sign of the intima or media. Release of the clamp at the level of the diaphragm yielded very poor back bleeding, even with good flow through the Gott shunt. Extension of the aortic incision into the abdomen finally revealed that the media from near the aortic isthmus had been inverted and intussuscepted down the descending thoracic aorta past the level of the diaphragm, which accounted for the unique arteriographic picture seen in Fig. 1. The media was then manually everted, and the intussuscepted aortic segment excised; it measured 12 cm in length, constituting the entire thoracic aorta distal to the left subclavian artery. A woven Dacron graft was then placed from the proximal descending thoracic aorta down to the intra-abdominal aorta just above the celiac trunk, which effectively replaced the thoracic aorta and obliterated the distal false lumen, but with the aortic clamps released, the patient was unable to sustain adequate cardiac output or systemic pressures and despite aggressive support with volume loading and inotropes, she died in the operating room from refractory myocardial failure.

Discussion

Traumatic rupture of the aorta due to blunt trauma is a frequent cause of death at the scene of the accident.[1] Although it is usually fatal, approximately 15% of the patients may survive beyond one hour after reaching the hospital,[2] and occasionally survive for several years with an unrecognized injury, although this is very uncommon.

With horizontal deceleration a tear occurs near the embryologic site of the ligamentum arteriosum; the key to survival is recognition of the widened mediastinum on an initial chest x-ray, as well as such other radiologic signs as elevation and rightward shift of the left mainstem bronchus, loss

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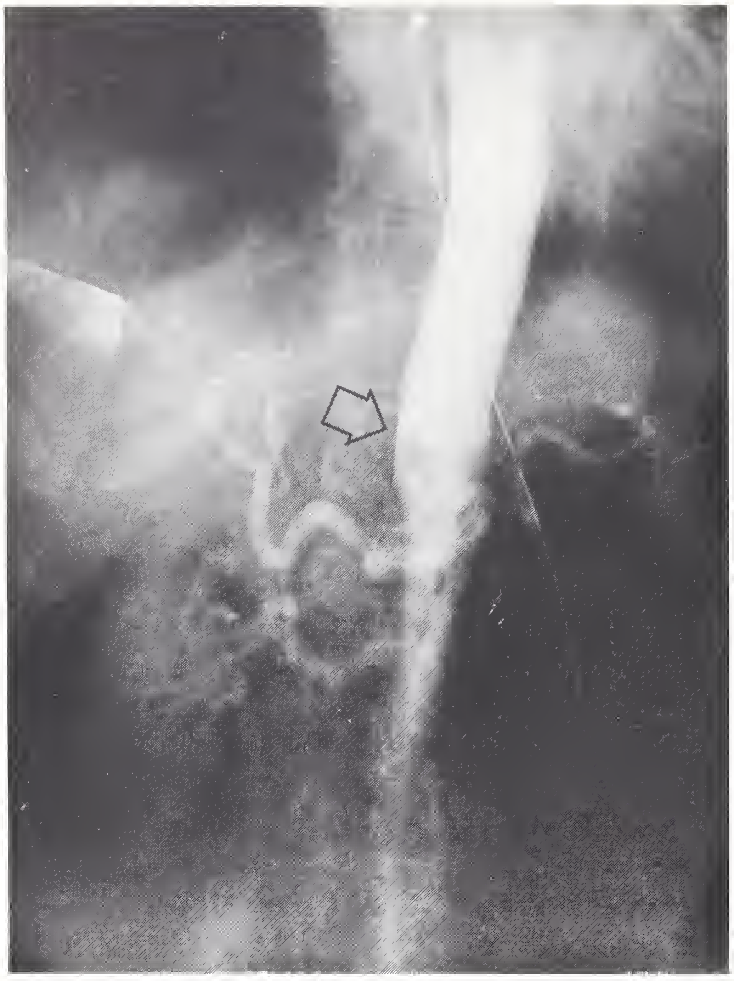

Figure 1. Transaxillary arteriogram with arrow showing cutoff of dye at celiac level and tapering of distal aorta. Left renal artery not visualized.

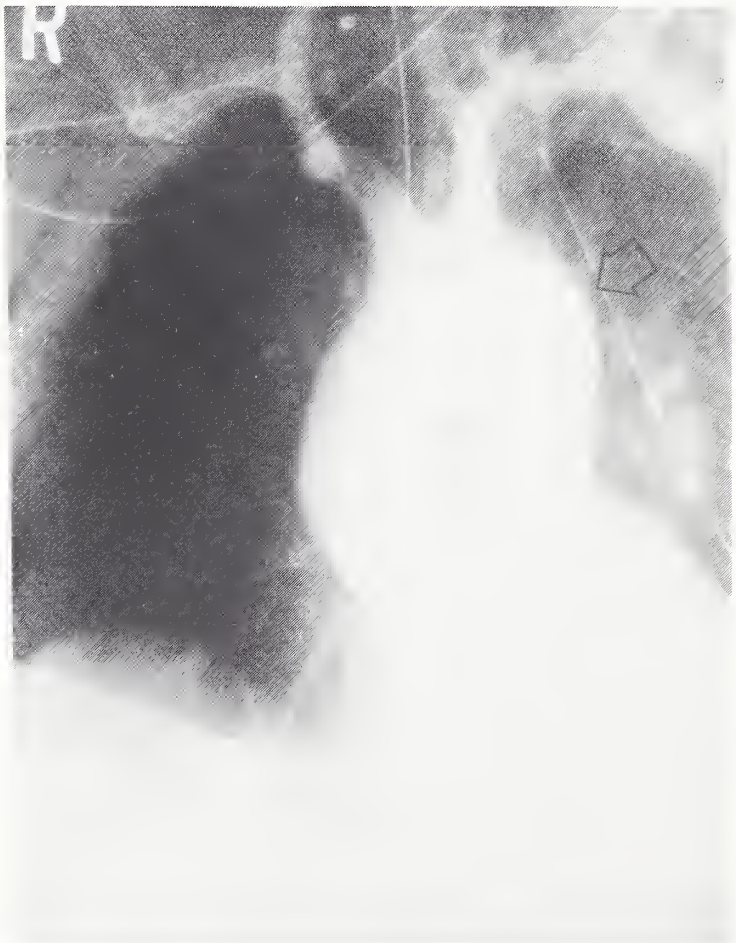

Figure 2. Arteriogram of aortic arch with arrow showing linear defect near site of proximal tear in aorta.

of the aortic arch silhouette, left upper lobe apical capping, and left first or second rib fractures. Definitive diagnosis is made by emergency aortogram. The femoral approach risks rupturing the false aneurysm, and as in our case may be misleading. The brachial approach, though associated with more catheter complications, probably gives more useful information. MacKenzie et al[3] recommended a supraclavicular incision to search for bleeding into the carotid sheath, but this is not widely accepted.

The injury is repaired through a left thoracotomy incision, and occasionally this may need to be extended to manage other injuries. There is some controversy over whether to shunt or not to shunt while the aortic graft is being placed. Many procedures have been used for shunting, including left heart bypass, aorta-to-aorta shunts, and left ventricle-to-aorta shunts.[4,5] The major complications in repairing these injuries are paraplegia, renal failure, left recurrent nerve palsy, and hypertension.[4]

On exploration, one usually finds a non-circumferential tear of the aorta, since most circumferential tears lead to exsanguination at the scene or en route to the emergency room. Circumferential tears that have been confined within the adventitia sufficiently long to reach the operating room may be separated by a distance of several centimeters.[6] The operative findings shown in our patient in which the aorta media and intima were stripped from the adventitia and inverted have not previously been described. Our patient's complaint of left leg pain was clearly related to ischemia of the left leg due to the intussuscepted media occluding flow to both the left leg and left kidney. Despite shearing of all intercostal arteries from the true lumen of the aorta, the patient was not paraplegic during her initial evaluation; one can only speculate about the likelihood of paraplegia had she survived the graft interposition.

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Radiology Case of the Month

SCOTT LOVELESS, M.D. and STEPHEN L. GAMMILL, M.D.

Case Report

A 56-year-old man had a four-day history of postprandial abdominal pain, described as sudden and sharp with radiation to his back. He denied nausea, vomiting or a change in stools. On examination he was afebrile and tender in the epigastrium, but had no masses or peritoneal signs. An abdominal CT with contrast is shown in Fig. 1. Please examine it and choose the best diagnoses. (There are two correct answers.)

(1) Pseudocyst
(2) Acute pancreatitis
(3) Pancreatic abscess
(4) Thrombosis of the superior mesenteric vein
(5) Pancreatic neoplasm

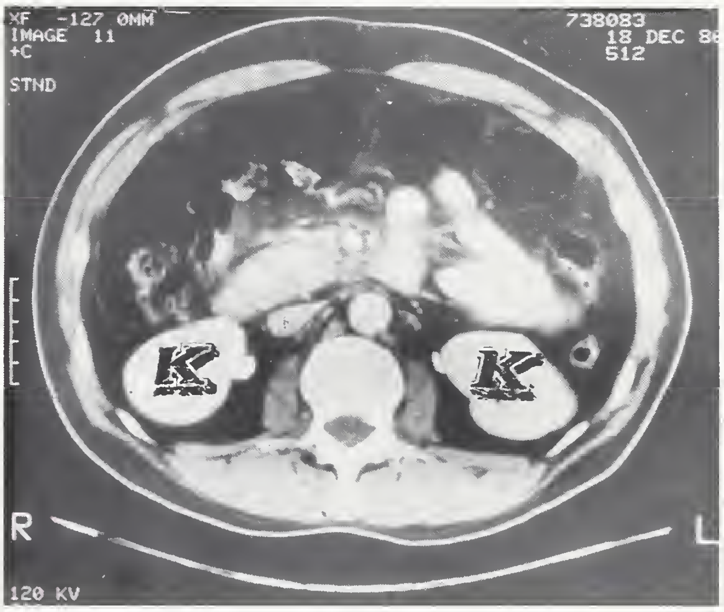

Figure 1. A cut through the pancreas at the level of the mid-kidneys (K). The inferior vena cava is visible with the head of the pancreas anterior to it. The superior mesenteric vein is adjacent to the pancreas near the midline and anterior to it.

Discussion

Pseudocysts. Pseudocysts are most commonly associated with subacute or chronic pancreatitis, representing a collection of necrotic tissue, old blood, and secretions that have escaped from the damaged pancreas. The term pseudocyst should be reserved for those fluid collections with a measurably thick fibrous capsule or wall. Pseudocysts may remain within the pancreatic capsule, but more commonly are found in an extrapancreatic location, usually in the lesser sac. They are detected by both CT and ultrasound in approximately 10% of patients with pancreatitis. On CT, pseudocysts are readily detected as unilocular or multilocular masses with a near-water density center and well-defined wall that occasionally calcifies. Complications of pseudocysts include hemorrhage, superinfection, and spontaneous rupture.[1,2]

Acute Pancreatitis. Most commonly due to alcohol ingestion or cholelithiasis, acute pancreatitis may present a broad spectrum of inflammatory changes ranging from mild edematous interstitial inflammation to fulminant necrotizing hemorrhagic pancreatitis. The CT appearance varies, showing a normal appearing gland in as many as one-third of patients. The abnormal CT findings of acute uncomplicated pancreatitis include diffuse or focal swelling of the gland, often with decreased attenuation values, and contour abnormalities with indistinct margins and increased density of peripancreatic fat planes. Morbidity and mortality associated with acute pancreatitis can be attributed largely to hemorrhage, pseudocyst, and abscess.[1,2]

Pancreatic Abscess. Pancreatic abscesses represent collections of pus and necrotic tissue within the pancreatic parenchyma, with potential extension into the lesser sac and retroperitoneum. They are usually the result of secondary infection of devitalized pancreatic and peripancreatic tissue following necrotizing pancreatitis. On occasion, pseudocysts become infected and are converted into abscesses. Although found in only 30% to 50% of proven abscesses, the most diagnostic CT finding is gas in the pancreas and surrounding tissues. More often, an abscess appears as a poorly defined inhomogeneous mass or fluid

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From the Department of Radiology, Baptist Memorial Hospital, Memphis.

collection, often with displacement of adjacent structures. The attenuation value is generally higher than that of sterile fluid collections or pseudocysts. Multiple abscesses occur in about one-third of patients, and are often far removed from the pancreatic bed.[1,2]

Superior Mesenteric Vein Thrombosis. The most common causes of mesenteric venous occlusions are inflammatory bowel conditions (peritonitis, abscess, inflammatory bowel disease), neoplasm (pancreas, colon), and blunt trauma. In the postoperative group, postsplenectomy patients were the most likely to develop superior mesenteric vein (SMV) thrombosis. Other less common causes of SMV thrombosis are cirrhosis, congestive splenomegaly, polycythemia vera, and migratory phlebitis. SMV thrombosis can occur as an isolated phenomenon or in association with portal and splenic vein thrombosis. Clinical findings vary and can be acute pain or slow, gradual onset of vague abdominal pain that increases in severity. Plain film findings are usually nonspecific. CT and angiography provide these diagnoses. The typical CT appearance is that of enlargement of the thrombosed SMV, a sharply defined vascular wall with a rim of increased density, and a central area of low attenuation representing clot within the vessel. The sharply defined, enhancing venous wall is thought to represent contrast enhancement by the arterially supplied vasa vasorum.[3,4]

Pancreatic Neoplasm. Adenocarcinoma represents 95% of the primary malignant tumors of the pancreas. Approximately 60% occur in the head, presenting early weight loss and jaundice. The remainder are in the body and tail, usually presenting later weight loss, back and abdominal pain, and occasionally a mass. The major CT finding is a focal soft tissue mass altering the contour of the gland. The density of the mass is usually similar to or slightly less than that of the normal gland. Other findings include obliteration of peripancreatic fat planes and loss of boundaries with surrounding structures, involvement of adjacent vascular structures and regional lymph nodes, pancreatic ductal dilatation, common bile duct obstruction, and hepatic metastases.[1,2]

Answer

In the case at hand, CT demonstrated the classically described findings of SMV thrombosis, with a hypervascular rim and central clot and indistinct tissue planes in the region of the uncinate process. Percutaneous needle biopsy showed cells suggesting malignancy, but subsequent exploratory laparotomy with multiple biopsies of the head and uncinate process revealed only pancreatitis.

FINAL DIAGNOSIS: Focal pancreatitis and superior mesenteric vein thrombosis.

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Loss Prevention Case of the Month

The Schedule vs the Patient

J. KELLEY AVERY, M.D.

Case Report

A 38-year-old man sustained a knee injury in a fall on the job at a construction site. He had a long history of alcohol abuse and peptic ulcer disease for which he had been seen regularly by his family physician. There was a strong family history of coronary artery disease in that the patient's father and an older brother had had coronary artery bypass surgery. Two weeks after his initial injury, the patient was admitted to the ambulatory surgery service at 7:00 AM scheduled for arthroscopy of the left knee at 10:30 AM. The physical examination done by the attending orthopedist five days prior to admission documented no positive findings except for those related to the left knee. The preoperative orders included a complete blood cell count, urinalysis, partial thromboplastin time (PTT), chest x-ray, and electrocardiogram. All the initial orders were carried out on the patient and, at the time, the anesthesiologist made his preoperative rounds. All results were reported on the chart and were within normal limits. During this visit, the patient told the anesthesiologist that he had some epigastric discomfort. The patient attributed this to his fasting state. The anesthesiologist ordered STAT cardiac enzymes at 9:30 AM. The blood was drawn for these tests at 9:40 AM, and at 10:00 AM the nurse administered the preoperative medication as ordered.

The operating surgeon arrived in the operating room about 10:40 AM and discussed with the anesthesiologist the cardiac enzymes that were not as yet reported. The patient denied any discomfort; the vital signs were stable, and both the anesthesiologist and the orthopedist thought that there was no good reason to delay the surgery. Within minutes after general anesthesia had begun, the patient suffered cardiac arrest, and attempts at resuscitation were unsuccessful. By this time the cardiac enzymes were reported and were strongly suggestive of an acute myocardial infarction.

A lawsuit was filed against both the orthopedic surgeon and the anesthesiologist.

Loss Prevention Comments

This young man had been followed and treated regularly by his primary care physician for complaints attributed to alcohol abuse and peptic ulcer disease. This was known to the orthopedic surgeon, but he made no effort to involve the patient's primary care physician in his plans for surgical treatment. The plaintiff position, supported by expert testimony, was that the orthopedic surgeon was negligent in not making contact with the patient's primary care physician regarding the past history and his plans for surgery.

The anesthesiologist promptly reviewed the patient's history during his pre-anesthesia visit and, because of the epigastric discomfort, ordered the cardiac enzymes STAT, assuming that the results would be reported before the scheduled surgery. He failed, however, to delay the preoperative medication until after the enzymes had been reported, and further, he agreed to begin anesthesia before the tests were reported. The plaintiff contended, again with the support of good expert testimony, that the anesthesiologist was negligent on both counts.

The orthopedic surgeon was accused of negligence for proceeding with the surgery before he had all the clinical evidence in hand with respect to the preoperative condition of his patient. No expert testimony could be obtained that the defendant physician had acted within the acceptable standard of care.

Other allegations complicating the defense related to the decision to operate in the ambulatory surgery area in view of the positive family history and in view of the continuing complaints related to peptic ulcer disease. The implication was that more attention was paid to cost containment, the surgical schedule, and the convenience of the physicians than to the welfare of the patient.

No good legal defense could be mounted; consequently, a large settlement was necessary.

Dr. Avery is chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood, and medical director for Ambulatory/Outpatient Services, St. Thomas Hospital, Nashville.

Health and Environment Report

Lead Poisoning in Children

FLORENCE B. ROBERTS, R.N., Ph.D.

Since antiquity, lead has been recognized as a dangerous substance, but recognition of the extent of the danger came slowly. Prior to 1960, a blood level of 60 μg/dl was not considered dangerous. In 1975 a level of 30 μg/dl was designated by the Centers for Disease Control as a level of requiring treatment. In 1985, the level was further reduced to 25 μg/dl.[1] Some researchers studying the subtle aspects of neurophysiology and behavior are now questioning the safety of that level.[2-4] In addition, there is recent concern for prenatal as well as postnatal exposure.[5]

The first federal legislation regarding lead was the 1971 Lead Based Paint Poisoning Prevention Act, which provided funding to assist the states in developing screening programs. That categorical program was folded into the Maternal and Child Health (MCH) Block Grant in 1981. In 1982, the Division of MCH urged that all children between the ages of 9 months and 6 years be screened at least annually.

Current Status

Lead poisoning affects approximately 4% of all children in the United States. It is not limited to children of any race or socioeconomic stratum, but poor black inner city children are at the highest risk. Of the southeastern states that have a lead screening program, the percentage of children 1 to 5 years of age with confirmed lead toxicity in 1983 ranged from 0.4% (South Carolina) to 1.5% (Arkansas). Of the states contiguous with Tennessee, Kentucky, Arkansas, North Carolina, Virginia, Missouri, and Georgia have statewide screening programs. Nationally seven states (Delaware, Illinois, Massachusetts, New Jersey, Rhode Island, South Carolina, and Wisconsin) have laws governing lead screening, and 13 others are considering such legislation.

Sources of Lead

Lead-based paint is still a major source of lead poisoning in children living in older housing. As a reflection of this hazard, Housing and Urban Development (HUD) advised in 1986 that all children under 6 years of age living in public housing built prior to 1978 should be screened for lead poisoning. In an environmental survey, HUD had found that nearly half of the housing units built prior to 1978 contained interior lead paint.

The reduction in use of leaded gasolines has somewhat decreased the hazard from airborne lead, but industrial airborne lead may still pose a threat in some locations. There is new concern for waterborne lead in older housing where lead solder was used on water pipes.

A common source of lead is dust carried home by workers in high lead industries, such as battery manufacturers. Some crafts, such as ceramics, use lead, which may present a hazard to the artist/craftsman or to children playing around their workspace.

Diets high in fat and low in calcium and iron appear to promote lead absorption. Nutritional counseling and increased access to dietary resources (such as WIC) may help to protect young children.

Screening

The screening test for lead is the erythrocyte protoporphyrin (EP) level. Children with an EP level above 35 μg/dl should have a blood determination for confirmation, since the EP reflects the degree of heme synthesis, and its determination is not a substitute for blood lead levels. Blood lead levels above 25 μg/dl are confirmatory of toxicity, and levels above 75 μg/dl indicate a need for immediate treatment.

At this time, except in Memphis, where the county has a small laboratory equipped to perform the tests for health department patients, both EP and blood lead determinations must be sent to private laboratories for processing.

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From the Tennessee Department of Health and Environment, Nashville.

president's page

James T. Galyon

One More Meeting

Most doctors I know look forward to the prospect of a new meeting with the same enthusiasm as a visit by the IRS. They already attend hospital staff meetings, specialty society meetings, committee meetings, and perhaps church and community meetings.

Recently I was a guest at a nearby medical society meeting. I was pleased to see a good attendance of physicians genuinely enjoying the company of fellow physicians. There was a sharing of problems, frank discussions, and development of plans that would hopefully meet these problems. Finally, there was a sound, scientific meeting presenting new treatment methods for a common medical problem. All of the ingredients of a good medical society meeting were there.

I thought about how unique a medical society meeting is. All of the other meetings tend to split doctors into groups. Hospitals are very competitive in their marketing and advertising, suggesting that their institutions are better than other hospitals, and by implication that their staffs are better than other hospital staffs. This creates a competitive environment between physicians that was not present a few years ago.

Specialty societies are wonderful means of broadening our knowledge in our limited field of medicine, but they also tend to set a specialty apart from others and even sometimes against other specialties.

Nowhere can one find the collegiality that extends across these lines of division like the local medical society. There you can work together to solve local problems and to use the political process on your local representatives at the various levels of government. There you can be informed about sociopolitical problems, which are evermore becoming a part of our daily practice of medicine. There you can also receive scientific information that will keep you abreast of the broad progress in medicine, and not just your own specialty.

By no means do I suggest that you go to your local medical society as a spectator only. We need involvement, even commitment, of all of our doctors. All of the other meetings have their place in our lives, but to leave out this one more meeting would be a serious error.

James T. Galyon, M.D

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JULY, 1987

editorials

A Tale of Two Chickens

Once upon a time people lived in castles, cottages, cabins, huts, or hovels, but no matter which, they shared one thing in common: their environment was drafty in winter and sweaty in summer. Most, even the castles, had animals under foot all the time, mostly for utilitarian purposes. Horses were ridden (if you were rich enough), cows were milked, dogs chased away footpads, and cats caught mice and rats, which were everywhere. If you wanted to eat a chicken, you (or if you were rich enough, your servant) reached out and caught one, wrung its neck, scalded it, plucked it, cooked it, and ate it—just like that. Tomorrow you ate tomorrow's chicken, since any of today's that was left had likely gone bad. Those were the (not so) good old days.

You've come a long way, baby. Though castles and hovels still tend to be drafty, most dwellings in between have gradually been improved to varying degrees by such things as storm windows and insulation to keep out both cold and heat, at least some of it. During a recent energy crunch the government placed a bounty on airtight houses. Homes are cleaner, too—or at least they can be; whether they actually are or not depends on personal habits. Nevertheless, nowadays if you want a chicken you go buy one that someone else has plucked, and maybe even cooked. It keeps a while, too, in the ol' fridge. Seems like heb'm, don' it? Well, it isn't.

Ever since he was ousted from Eden, man, along with his significant other(s), has been looking to regain it on his own. Many of the blessings he has manufactured along the way have turned out to be not unmixed. Cited above are two of them. Tighten up your house and radon gas may accumulate and give you lung cancer. Then too, if you tightened it up with asbestos, you got a double dose. The manufacturers who used asbestos in making insulation went bankrupt. Their reorganized companies took to using glass fibers, which some epidemiologist now says is also carcinogenic.

Back when chickens were plucked, cooked and eaten on the spot, if the finished product had taken on a little guano in the process, it was of small moment, as the dose of coliforms was not much. These days most chicken feet never touch the ground. Chickens are raised and slaughtered in a controlled environment, and presumably reach you clean—presumably, because the incidence of gastrointestinal illness due to *Salmonella* and other enteric pathogens from eating guanoed poultry is skyrocketing as the cognoscenti eschew chewing red meat in favor of fish and chicks—and just when it looked as if we civilized types were about shed of the lowly bacterium, as well as most viruses, as a threat to our life, liberty, and pursuit of happiness (LLAPOH—pronounced llaphoh). When Mr. Roosevelt promised two chickens in every pot and a car in every garage, he didn't have this in mind, but then the death and burial of most of those chickens would have been close to simultaneous.

There are not very many things around about which there is not both good news and bad news. Litigiousness over the bad news has diminished enthusiasm for going after the good, witness the disinclination of biological manufacturers to make vaccines.

When I was growing up on Lookout Mountain a dirty pall obscured the valley below, in which Chattanooga was hiding. During my college and medical school days the same soft coal product shrouded objects on the opposite side of Nashville's streets, and turned daylight into dusk. Natural gas was the answer, but supplies were finite, and the real answer was to be atomic energy—until we had it. The good news was that it indeed was clean, efficient, and available. The bad news was that, like any animal, it produced waste—radioactive waste. Not only that, it was radioactive waste with a long half-life. What to do with it, as well as with toxic wastes from chemical processing plants, has occupied a lot of time and energy in the Congress and elsewhere, without any satisfactory results.

The list could go on and on, but I'm getting writer's cramps, and you will stop reading, anyway. If this story has a moral, it is that we always expect too much, which is not necessarily bad, as it tends to push toward a solution. When those expectations become demands, however, and when society exacts punishment for failure to meet those demands over and above the failure itself, initiative falters and productivity dwindles. Worse, the bureaucrats get into the act, and both diminish productivity further and drive up the cost. There is no way, for example, to discover the contaminated chickens by quick screening. It requires *minute* examination (by bacteriology, I presume) on random specimens to find where the culprits are coming from.

And so, in our search for absolute security the bureaucracy proliferates—but at what a price! Things move best in a laissez-faire environment; but this is also at a price—caveat emptor. To make society work requires a fine balance. Imbalance, however, is the rule; the balance tends to fluctuate rather than remaining in balance. These days bureaucracy is in the saddle, and medicine is caught in its trap.

J.B.T.

A More Perfect Union

. . . To insure domestic tranquility, . . . promote the general welfare, and secure the blessings of liberty to ourselves and our posterity . . .

Preamble to the Constitution
of the United States

I had thought to write something about the Constitution of the United States to mark its bicentennial this year, but during preparation for it I found the subject too depressing—not the Constitution, which is marvelous in its simplicity and directness, and for which I have the highest regard, but the courts and the manner in which they have distorted it, which isn't and for which I don't. The courts' "business" generally was not their business in the first place. The three branches of government were set up in such a manner as to balance each other and protect the citizenry from excesses of any one branch. It was a good plan, one that would secure for us the blessings of liberty that were won at such cost. In practice, the citizenry has no protection against the vagaries of the unelected, frequently tyrannical presidential appointees manning the federal benches. Hence the citizenry, instead of enjoying the fruits of the Revolution, simply traded King George III for the Supreme Court, which is free to impose upon us its own political and social philosophies without redress.

Since federal judges are appointed by the President for so long as they behave themselves, and since they presumably reflect his views—though some, once appointed, prove renegades—a long-term president, such as Mr. Roosevelt, for instance, can influence the nation for generations to come. No one knows how to abuse power like a lawyer; Jesus pronounced woes on them because they were blind guides who inflicted burdens too difficult to bear, and He accused them of straining out gnats and swallowing camels.

According to the Constitution, legislative power belongs to the people through their elected representatives, but federally only as much as specified. Actually, few things are delegated to the federal government. "The powers not delegated to the United States by the Constitution, nor prohibited by it to the States, are reserved to the States respectively, or to the people [through the process of amendment to the Constitution]." (Amendment X.)

The tenth amendment, which was required by the States because of early abuse of federal power, has been almost universally violated from the first.

The Golden Rule says that "he who has the gold makes the rules." It became necessary for the federal—not the state—government to have the gold because of the demands by the people for a place at the federal teat. And so the public, like Esau, traded its birthright for a mess of potage. This originated long before the reign of Mr. Roosevelt, but he did help it—or rather propel it—along.

Instead of celebrating the bicentennial of the Constitution, I think we should be holding a wake. It is not a "living document," as some jurists insist, but only an ossified instrument that the courts can—and do—use on us like the jawbone of an ass in whatever way suits their fancy. But the major culprit is not, as popularly held, the Supreme Court or even the bureaucracy. Though it was the courts that delivered the mortal blow (actually a series of them beginning soon after the Supreme Court was formed), neither they nor anyone else in Washington killed it. The Constitution was killed by "We, the people of the United States," who just stood around and watched it die. In order to form a more perfect union (we thought), we sold out justice, domestic tranquility, the general welfare, and the blessings of liberty—for ourselves and our posterity.

We have secured a union, all right, but what we have secured is not an amalgam; it is more anodized, where only the all encompassing federal power structure counts. Beneath the shiny outsides is the nondescript mass of white metal that serves only to maintain mass and shape, and lend support. That's us! But as Major Pickering said in *My Fair Lady*, "We-e-e didit!"

The Founding Fathers must be spinning in their graves.

J.B.T.

Stultification Simplified

Spiderman, affectionately known to aficionados as Spidey, is getting married—not once, but three times! You see, there is Spiderman, and there is Spiderman; and then, there is Spiderman. One is the property of Marvel Comics, makers of comic books; another belongs to the syndicated daily strips. The third is sort of free lance; he will be married in person in Shea Stadium on June 5. The other two will be married around the same time in their respective media, but in different ways, though each to the same girl—fortunately, I guess. It is tidier that way—and also legal.

Reaction to the coming nuptials is varied. One 19-year-old monitor of a store's comic book section thinks getting married is "old and boring. It looks like they could do something more interesting," he said.

My own relationship to Spiderman is tenuous at best, since he belongs to another generation. Mine is Terry and the Pirates and Buck Rogers (also Will); it is of a time where to be properly sanctioned, a relationship had to wind up before an altar or its surrogate. Now it is OK just to have a relationship; that is, it is in circles other than the ones I move in, which are "old and boring," I guess. I guess I know too what he meant by something more interesting, though what he said was, "They could have dyed their hair yellow." Not that all the relationships in my day—or any other day that I know anything about—actually *did* wind up sanctioned, but it was at least looked upon as the thing to do; in fact, not to was actually illegal—maybe still is, though any test case would doubtless be thrown out of court as a violation of constitutional rights. (It seems almost everything that promotes what we used to call decency violates somebody's constitutional rights. But I digress.)

Now, back to "old and boring." Spoken like an unmarried nit. I think, though, that what he actually meant was not "boring," but "a bore," referring to the concept; the distinction would doubtless be lost on him. Given today's moral climate, it does not surprise me he would think that, though it does disappoint me. He doubtless is thinking, as men (maybe women, too) have since Adam, of all the wild oats left him to sow. It is just that it is now acceptable to express one's boredom with it.

The news media are continually—no, continuously—preoccupied with something or other, often to the point of beating a dead horse. They continue beating that horse until the next live one comes along. It usually has to do with somebody or other's peccadillos, since those are the jucier morsels that sell newspapers and lure tube-watchers. Sometimes they get a bonanza—two at once. Crowding out Jim and Tammy Bakker, and even Iran-gate for a while, were the comings and goings of the late lamented (?) presidential candidate Gary Hart, et al, who early in the game shot his candidacy in the foot. Whether or not he (or rather his candidacy) was a victim of the media or simply its patsy depends on whom you ask. Without taking sides in the dispute, the *Wall Street Journal* observed that this preoccupation reflected a certain poverty of ideas, and it was that that bothered them most of all.

In Athens the Apostle Paul found the men of the city assembled in the Agora, as was their daily wont, to see and to hear some new thing. The first observation one would have to make is that they had not enough to do. That would necessarily be followed by the observation that they could be doing worse things. Boredom does not, after all, depend, or at least depend exclusively, on external circumstances. Boredom is an inner lesion.

Spiderman's marriage, whenever or however, does not strike me as culturally enriching or mentally stimulating. Nonetheless, it does not bother me that our adolescent philosopher might find it so, or that he would consider marriage "old and boring." Neither does it bother me much that he might consider their dyeing their hair yellow an interesting alternative. What does bother me is the suspicion that he has a lot of company, and that in a cokey funk they might consider the interesting alternative painting the town instead. Even worse is the knowledge that many in his company are not adolescent, and may be in positions of authority.

The media have no corner on poverty of ideas. Mental impoverishment is far more prevalent than financial poverty. It is also more dangerous, and even harder to cure. Either way, it appears the poor we have with us always.

J.B.T.

TMA Members Receive AMA Physician's Recognition Award

Forty TMA members qualified for the AMA Physician's Recognition Award during April 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Carolyn F. Blackwell, M.D., Memphis
Robert R. Cleveland, M.D., Murfreesboro
Thomas S. Dake, M.D., Columbia
Phillip H. Dirmeyer, M.D., Memphis
John H. Dougherty, M.D., Fairfield Glade
William H. Fancher, M.D., Memphis
Gordon R. Freeman, M.D., Dyersburg
Francis W. Gluck, Jr., M.D., Nashville
James A. Greene, M.D., Knoxville
Charles N. Hatfield, M.D., Maryville
William H. Hill, M.D., Madison
Robert P. Hornsby, M.D., Murfreesboro
Wesley F. Jones, Jr., M.D., Lexington
Eugene R. Kidwell, Jr., M.D., Kingsport
Fred T. Kimbrell, Jr., M.D., Nashville
Rodger P. Lewis, M.D., Union City
William E. Long, M.D., Memphis
William A. Loy, M.D., Oak Ridge
William S. McDougal, M.D., Nashville
Charles A. Mitchell, M.D., Nashville
Lawrence R. Nickell, M.D., Columbia
Pamela T. Osborne, M.D., Cordova
William T. Patten, M.D., Hendersonville
Andres A. Ramos, M.D., Gallatin
Larry D. Reed, M.D., Crossville
Leslie B. Reynolds, Jr., M.D., Kingsport
Samuel S. Riven, M.D., Nashville
Randall G. Samples, M.D., Cookeville
Glen C. Shults, M.D., Newport
Langdon Smith, M.D., Nashville
Russell R. Smith, M.D., Goodlettsville
Clarence L. Spannuth, M.D., Johnson City
Naveen Srinivas, M.D., Nashville
Robin M. Stevenson, M.D., Memphis
Brian R. Swenson, M.D., Nashville
Lynn B. Tepley, M.D., Chattanooga
Lloyd R. Thomas, Jr., M.D., Memphis
David T. Upchurch, M.D., Oak Ridge
Walter W. Wheelhouse, M.D., Springfield
William G. Wood, M.D., Johnson City

in memoriam

Richard Baker Austin, III, age 71. Died April 9, 1987. Graduate of Tulane University School of Medicine. Member of Montgomery County Medical Society.

James S. Bell, age 49. Died April 30, 1987. Graduate of University of Tennessee College of Medicine. Member of Memphis-Shelby County Medical Society.

Don E. Dismukes, age 60. Died April 30, 1987. Graduate of University of Tennessee College of Medicine. Member of Memphis-Shelby County Medical Society.

Robert Mark Finks, age 74. Died May 22, 1987. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

James C. Gardner, age 86. Died April 28, 1987. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

Henry Thomas Slawson, Jr., age 62. Died February 18, 1987. Graduate of University of Tennessee College of Medicine. Member of Memphis-Shelby County Medical Society.

Wendell B. Thrower, age 61. Died May 9, 1987. Graduate of Medical University of South Carolina. Member of Franklin County Medical Society.

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

KNOXVILLE ACADEMY OF MEDICINE
John M. Ambrosia, M.D., Knoxville
John W. Mack, Jr., M.D., Knoxville

MEMPHIS-SHELBY COUNTY MEDICAL SOCIETY
William Kearney Cook, M.D., Memphis
Mark Lane Cunningham, M.D., Memphis
Barbara A. Duncan-Cody, M.D., Memphis
Robert Lewis Jackson, M.D., Memphis
Edward Patton Scott, M.D., Memphis
James Carroll Varner, M.D., Memphis
Julian Mack Worthington, M.D., Memphis

NORTHWEST TENNESSEE ACADEMY OF MEDICINE
Marquerite McIntosh, M.D., Newborn

ROANE-ANDERSON COUNTY MEDICAL SOCIETY
David Dale Long, M.D., Oak Ridge

RUTHERFORD COUNTY/STONES RIVER ACADEMY OF MEDICINE
Mark L. Born, M.D., Murfreesboro

WHITE COUNTY MEDICAL SOCIETY
Alan Drake, M.D., Sparta

personal news

Donald R. Lechler, M.D., Chattanooga, has been certified as a Diplomate of the American Board of Anesthesiology.

announcements

CALENDAR OF MEETINGS

NATIONAL

Aug. 14-19	Association of Medical Illustrators—Radisson South, Minneapolis
Aug. 28-Sept. 2	World Conference on Health Education—Houston
Sept. 9-12	American Society for Surgery of the Hand—Convention Center, San Antonio, Tex.
Sept. 12	American Rhinologic Society—Las Vegas
Sept. 14-17	American Academy of Family Physicians—Fairmont, San Francisco
Sept. 14-19	American Electroencephalographic Society—Adam's Mark Hotel, St. Louis
Sept. 16-19	American Thyroid Association—J. W. Marriott, Washington, D.C.
Sept. 18-19	American College of Nuclear Medicine—Shoreham Hotel, Washington, D.C.
Sept. 19-23	American Academy of Otolaryngology-Head and Neck Surgery—Chicago
Sept. 22-24	American Paraplegia Society—Riviera Hotel, Las Vegas

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Tuberculous Peritonitis

Case Report and Review of the Literature

DOUGLAS A. GENTILE, M.D. and CHARLES B. ROSS, M.D.

Tuberculous peritonitis (TBP) is a readily treatable disorder that is frequently overlooked in the differential diagnosis of ascites. Several factors contribute to the difficulty in making a clinical diagnosis of TBP. The disorder is uncommon, has a variable and nonspecific clinical presentation, and often occurs in the absence of tuberculosis clinically apparent elsewhere. This applies particularly to the patient with coexisting TBP and alcohol-induced cirrhosis. Even when TBP is clinically suspected, its confirmation may prove difficult. We present a case recently seen in our hospital and review the epidemiology, clinical manifestations, diagnostic evaluation and therapy of TBP.

Case Report

A 21-year-old black woman came to the Vanderbilt University Medical Center Emergency Department with a three-week history of progressive abdominal distention, nausea, early satiety, weakness, low grade fever, and dyspnea with four pillow orthopnea. She denied having night sweats, chills, or weight loss. She had been in good health until the onset of this illness, without past medical history of exposure to tuberculosis; she stated that she had had negative tuberculin skin tests in school. She did not smoke or use alcoholic beverages, she reported normal menses, and denied previous sexual intercourse.

On physical examination, the patient appeared ill, with marked abdominal distention and tachypnea with shallow inspirations. Her temperature was 101.2°F orally, pulse rate 144/min, blood pressure 144/98 mm Hg, and respiratory rate 28/min. Her skin was without lesions and there were no abnormal findings on HEENT examination. There was no palpable adenopathy. Auscultation of the chest revealed bibasilar rales with diminished breath sounds at the right base. The abdomen was nontender and without palpable masses, but was tensely distended, with a positive fluid wave. Pelvic and rectal examinations revealed no pathology. There was bipedal edema. Neurologic examination was within normal limits.

Admission laboratory studies revealed a WBC count of 5,000/cu mm with a differential of 65% segmented neutrophils, 14% band forms, 14% lymphocytes, 5% monocytes, and 2% eosinophils. The hemocrit was 29.6% (hypochromic, microcytic), and the platelet count was 609,000/cu mm. The erythrocyte sedimentation rate (Westergren) was 51 mm/hr (normal 0 to 20 mm/hr). Serum electrolytes, creatinine, coagulation profile, serum beta-HCG, and a urinalysis were all within normal limits. The serum albumin, total protein, SGOT, alkaline phosphatase, and total bilirubin were normal. The LDH was 345 U/L (normal 125 to 250 U/L). A chest x-ray revealed a moderate right pleural effusion. An abdominal series revealed no free intraperitoneal air but suggested a large ascites, confirmed by abdominal-pelvic ultrasound and computed tomographic scans.

Abdominal paracentesis on the night of admission yielded 1,250 cc of cloudy, yellow fluid with 3,600 RBCs/cu mm, with 1,450 WBCs/cu mm, 82% mononuclear cells, and 18% segmented neutrophils; glucose was 79 mg/dl. (The serum glucose was 86 mg/dl; ascitic/blood glucose ratio was 0.92.) The peritoneal fluid protein was 5.5 gm/dl, LDH 388 U/L, and amylase 26 U/L. Bacterial cultures as well as acid-fast bacilli (AFB) smears and cultures ultimately were negative. An as-

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From the Division of Trauma, Emergency Service, Vanderbilt University School of Medicine, Nashville.

Reprint requests to 1367 Emergency Department, Vanderbilt University Hospital, Nashville, TN 37232 (Dr. Gentile).

citic fluid cell block contained reactive mesothelial cells. A right-sided thoracentesis revealed 8,400 RBCs/cu mm and 2,050 WBCs/cu mm (82% mononuclear cells, 18% segmented neutrophils); protein was 5.25 gm/dl and glucose 91 mg/dl, and bacterial and AFB cultures were ultimately negative. Multiple blood, sputum, and urine cultures were negative. A 5-TU intradermal PPD was placed and was strongly positive at 48 hours with induration and erythema over an area of 20×27 mm. AFB smears and cultures of the sputum were negative.

Exploratory laparotomy performed to differentiate between malignant disease and TBP disclosed peritoneal and serosal surfaces studded with implants and the small bowel matted with multiple interloop adhesions without evidence of obstruction. The small bowel mesentery was thick and shortened and the omentum was described as "cake-like," biopsies of which revealed caseating granulomas with a single acid-fast bacillus.

Ascitic fluid and tissue specimens for AFB cultures were obtained, and an antituberculous drug regimen was started consisting of isoniazid (INH), ethambutol, rifampin, and pyridoxine; when the patient developed elevated SGOT and alkaline phosphatase, the rifampin was discontinued. Except for a urinary tract infection that responded to treatment with ampicillin, the postoperative course was uncomplicated, and on Aug. 29, 1985, the patient was discharged from the hospital on doses of INH 300 mg/day, pyridoxine 50 mg/day, and ethambutol 15 mg/day. AFB cultures of the sputum, ascitic fluid, and pleural fluid showed no growth, but culture of the peritoneal tissue yielded *Mycobacterium tuberculosis*, sensitive to INH, ethambutol, and rifampin. Following discharge from the hospital, the patient improved considerably and remained compliant with her medications; she had no further febrile episodes and currently has no evidence of ascites.

Discussion

TBP is an uncommon disorder. Sochocky[1] reported an incidence of less than 0.5% among patients admitted to tuberculosis sanitoria between 1953 and 1966. Alvarez and McCabe[2] found the same percentage of TBP in patients hospitalized for tuberculosis between 1968 and 1977, which corresponds well with the rate of 0.43% of total cases of tuberculosis reported nationwide for the period 1969 to 1973.[3] There were 21,701 cases of tuberculosis reported in the United States in 1984.[4] Assuming an incidence of 0.5%, one would expect approximately 100 new cases of TBP per year nationwide.

Although TBP is relatively rare, certain groups are at higher risk; it is more common in nonwhites[1,5-7] and has classically been found more commonly in women,[1,6,8,9] although recent series report a nearly equal sex distribution.[2,5,7,10] The incidence is particularly high in two populations: black women [8,11,12] and alcoholics.[2,5,10] The referral area for a hospital appears to determine the epidemiologic pattern of the disorder. If the referral area is large, the majority of patients tend to be young, black women; if it is limited to a large urban area, alcoholics will predominate, without racial or sexual predilection.[1]

The pathogenesis of TBP has been widely discussed, and probably involves at least three separate mechanisms. Peritoneal involvement may develop during the bacteremia of miliary tuberculosis.[8,10] This probably represents only a small portion of cases, however, as miliary TB is not present in most patients with TBP. The peritoneum may also become infected via direct extension from contiguous structures. Tuberculous involvement of the female genital tract is well documented,[13] and in the past was believed to be the most common source of peritoneal seeding. Karney et al[5] found culture-proven pelvic tuberculosis in 30% of women with TBP, but for reasons that are unclear, two other recent large series did not find any evidence of pelvic tuberculosis.[9,10] There may be direct extension to the peritoneum from tuberculous intestinal ulcerations, although coexisting enteritis and peritonitis are uncommon.[2,6,8-10,14]

The most common mechanism for the development of TBP is hematogenous spread to the peritoneum during primary pulmonary infection. This may occur as a "silent" bacteremia, resulting in the formation of healed peritoneal tubercules that can later reactivate without any evidence of pulmonary involvement.[2,9,15] Alternatively, peritonitis may accompany active, culture-proven pulmonary TB as part of a disseminated tuberculosis.[5,8,14,15] As in this case, the pulmonary involvement is usually of the primary type.[8]

TBP typically has an insidious onset. In the usual case, patients suffer symptoms for three months before definitive diagnosis,[1,5,8-10] but symptoms may develop suddenly and patients may even have clinical signs of an acute surgical abdomen.[1,10,16] Almost all patients complain of constitutional symptoms such as anorexia, weight loss, malaise, weakness and fatigue. Increased abdominal girth and/or distention are complaints in 50% to 60% of patients, and the majority complain of abdominal pain.[2,5-8,10,11,14] Fever is present in 80% to 100% of patients, although it may be intermittent and is often of low grade.[2,5-10] Ascites and abdominal tenderness are frequent (70% to 100%)[2,5-10] and an abdominal mass or hepatosplenomegaly is observed on occasion (20% to 30%).[2,5-7,14] The classic "doughy abdomen" is very uncommon in modern series, and probably represents a late phase of the disease.[1,5,8,10,17]

Routine laboratory and x-ray studies are often abnormal but rarely diagnostic. The WBC count is usually normal, although it may be either increased or decreased. While there may be an increase in polymorphonuclear cells, monocytosis is not typical. The hemoglobin is usually normal. Liver function tests often show minor abnormalities, but are not characteristic.

The chest x-ray is often normal, but may suggest tuberculosis, as it did in this patient. A pleural effusion is found in 30% to 50% of patients, but parenchymal infiltrates occur in only 5% to 15%.[6,8,9,14,17] The 5-TU PPD is usually positive,[2,6,9,14] although series from this country have reported negative PPD tests in as many as 70% of patients with TBP[5]; a negative test appears particularly likely in the alcoholic patient.

Peritoneal fluid analysis is essential in the evaluation of a patient with ascites, but is rarely diagnostic of TBP. Unlike tuberculous pleural effusions, ascitic fluid is rarely bloody.[5,8,10] The protein content is characteristically greater than 3.0 gm/dl[2,6,8,9,14] but may be less, particularly in alcoholic patients with cirrhosis.[2,5,10] The leukocyte count is usually greater than 250/cu mm, with predominantly mononuclear cells, but it may be lower in up to 30% of patients,[2,10] and polymorphonuclear cells may predominate early in the course of the disease.[2,5,10,18] Culture of the peritoneal fluid is frequently positive for *Mycobacterium tuberculosis,*[9] but is of little help in attaining diagnosis in acute disease due to the long incubation period. Acid-fast stains are rarely positive.[6,9,14]

The glucose concentration in ascitic fluid is often low in TBP.[19] In particular, it has been suggested than an ascitic/blood glucose ratio of less than 0.96 may help differentiate patients with ascites due to TBP from those with ascites due to cirrhosis,[20] but low ascitic glucose levels may also be found with peritoneal carcinomatosis and other inflammatory disorders.[21]

Additional diagnostic modalities include abdominal CT and gallium scan. CT may show features suggestive of TBP, such as high density ascites or irregular soft tissue densities in the area of the omentum (not found on this patient's CT scan).[22,23] Gallium scan shows accumulation of gallium in the abdominal cavity.[24,25] Neither test is diagnostic.

From the preceding, it is apparent that the clinical diagnosis of TBP may be difficult. It is an uncommon disorder that may simulate a number of abdominal illnesses, and laboratory and x-ray findings may be nonspecific. This is a particularly difficult problem in alcoholic patients with TBP. Ascites in these patients is often attributed to cirrhosis, and their symptoms may mimic alcohol-induced hepatitis. The diagnosis is not infrequently made at autopsy.[6,7,10,14]

A caseating granuloma or AFB on stain is usually required to make the diagnosis of TBP, and in older series, this often meant exploratory laparotomy.[6,7,10] The diagnosis is still often made at laparotomy, either because another diagnosis is initially suspected[2,11,14,16] or because other less invasive studies are unrevealing.[5,8,9] In complicated cases, such as those with intestinal obstructions or when the risk of perforation is deemed to be high, exploratory laparotomy may be the initial procedure of choice.

Peritoneal biopsy with a Cope needle is an alternative that is often diagnostic. As described by Levine,[17] a puncture is made in the left lower quadrant level of the rectus sheath, through which three to four bits of peritoneum are obtained from different quadrants of the same puncture site. In a large prospective study by Singh et al,[9] the diagnosis of TBP was made without complications in 30 of 47 (64%) patients with this technique, but hemorrhage, small bowel perforations, and death have been reported.[14,26] In general, it should be attempted only if there is free ascitic fluid and no interfering abdominal mass is palpated.

Peritoneoscopy with biopsy is another technique that may be useful where equipment and trained personnel are available.[14,27,28] It allows for direct visualization of the peritoneum and abdominal structures, with biopsy of involved areas. In the study by Singh,[9] 10 of 17 patients who had negative Cope needle biopsies had a positive biopsy obtained by peritoneoscopy, while the remaining seven required laparotomy for diagnosis.

TBP in the pre-chemotherapy era was frequently fatal,[10,29] but with good patient compliance, cure rates approach 100%.[1,2,5,8,9] There are no controlled studies comparing single or multiple antituberculous agents in TBP. Generally, isoniazid in combination with one or two additional drugs is employed for 18 to 24 months. Dutt et al[30] have recently reported their experience with short-course chemotherapy for extrapulmonary tuberculosis. Peritoneal tuberculosis was found in 17 out of 402 cases. For drug-susceptible disease, treatment consisted of isoniazid 300 mg and rifampin 600 mg daily for one month, followed by isoniazid 900 mg and rifampin 600 mg twice weekly for another eight months. They reported an overall success rate of 95% in patients who completed therapy. The use of corticosteroids has been advocated to prevent late fibrotic complications, such as bowel obstruction from adhesive bands. Although there are no controlled studies, their use may be beneficial.[9]

Summary

TBP is an uncommon but important cause of ascites, particularly in young patients and alcoholics. Diagnosis usually requires a peritoneal biopsy demonstrating caseating granulomas or AFB. It is a readily treatable disorder, and therefore an important one to consider in the differential diagnosis of ascites.

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Maternal Death Due to the HELLP Syndrome

MICHAEL S. CARDWELL, M.D.

Preeclampsia-eclampsia is one of the five leading causes of maternal mortality in the United States.[1] Although much is known about its diagnosis and treatment, the etiology is still to be elucidated. Severe preeclampsia has been described as "another great imitator."[2] The presentation may be atypical and a timely diagnosis may not be made. The following case describes a maternal death due to the HELLP syndrome (hemolysis, elevated liver enzymes and low platelets), a severe variant of preeclampsia-eclampsia which presented atypically.

Case Report

The patient was a 35-year-old gravida 4, para 2, abortus 1 white woman with an EDC of Nov. 1, 1986. Her previous pregnancies were unremarkable. She has no history of significant medical illnesses and specifically no history of hypertension or chronic renal disease. For the past two years she had intermittent right upper quadrant pain thought to be due to mild gallbladder dysfunction which was treated symptomatically.

On Aug. 17, 1986 the patient awoke with severe right upper quadrant pain associated with nausea. The pain was similar to her other "gallbladder attacks," but more severe. The patient came to the emergency department at 7:35 AM at a gestational age by dates of 29-3/7 weeks. Physical examination revealed a tender epigastrium and right upper quadrant pain to palpation. There was no jaundice or edema; temperature was 98°F, pulse 80/min, respirations 32/min, and blood pressure 160/80 mm Hg. Hemoglobin and hematocrit were 15.7 gm/dl and 46.1%, WBC count 14,100/cu mm with 34% segmented neutrophils and 55% lymphocytes; platelets were adequate, and electrolytes normal; amylase was 128 U/L and the SGOT 617 U/L. Urinalysis was positive for blood, negative for bilirubin, and 4+ for albumin. The general surgery consultant thought the clinical picture was atypical for cholecystitis but ordered an ultrasound of the gallbladder. The sonogram revealed thickening of the gallbladder wall and a solitary stone. The patient was transferred to the delivery suite for further evaluation, where fetal heart tones were in the 110 to 160 range, and repeat blood work showed an SGOT of 5,680 U/L and a total bilirubin of 4.5 mg/dl and direct 1.9 mg/dl. The WBC count was now 27,400/cu mm with 53% band forms, 37% segmented neutrophils, and 6% lymphocytes. The platelets appeared adequate; the serum was red.

At 3:00 PM the patient complained of epigastric pain and a headache; her blood pressure at 3:30 PM was 148/112 mm Hg, and her headache was worse. The nurse was unable to arouse the patient at 4:30 PM, and shortly thereafter the patient had a generalized seizure. Her blood pressure postictal was 200/100 mm Hg and she was unresponsive to any stimuli; decerebrate posturing was evident. Fetal heart tones were lost during the seizure and intrauterine fetal death was documented with real time ultrasound at bedside.

A neurosurgical consultant made a diagnosis of massive intracranial bleeding; bleeding was now present from all previous venipuncture sites, and grossly bloody urine was flowing from the bladder catheter. A CT scan of the head revealed multiple collections of blood intracranially and a possible sagittal sinus thrombosis. The CT scan of the abdomen was consistent with a large clot in the inferior vena cava. Laboratory evidence indicated disseminated intravascular coagulopathy (DIC); platelet count was 133,000/cu mm, prothrombin time 23.5 seconds, partial thromboplastin time 81 seconds, fibrinogen 34 mg/dl, and fibrin degradation products greater than 40 mg/L.

The patient was transferred to the neurosurgical intensive care unit and placed on life support systems. Fresh frozen plasma, platelets and blood were given to correct the coagulopathy, and evacuation of the uterus with vaginal prostaglandin E2 suppositories was initiated in order to terminate the DIC. In spite of these measures, the patient's condition deteriorated and life support was withdrawn after cerebral nuclear flow studies and serial EEGs demonstrated no cerebral function.

Autopsy showed extensive intrahepatic hemorrhage and necrosis, cerebral edema, bilateral pulmonary atelectasis, bilateral pleural effusions, and epicardial petachiae, as well as thrombosis of the sagittal, transverse and sigmoid sinuses, a left subdural hematoma, and multiple cutaneous ecchymoses.

Discussion

The HELLP syndrome, a severe variant of preeclampsia-eclampsia, was first reported as a separate entity by Weinstein in 1982 from a series of 29 patients who had severe preeclampsia with hemolysis (H), elevated liver enzymes (EL), and low platelets (LP).[3] Our patient had hemolysis as evident from the red serum and bloody urine, elevated SGOT and bilirubin, reflecting massive hepatocellular damage, and a platelet count of 133,000/cu mm, grossly abnormal for pregnancy.[4] Her initial blood pressure was slightly elevated at 160/80 mm Hg, but she was in pain and apprehensive. Only 13 of Weinstein's 29 patients had an admission blood pressure higher than 160/110 mm Hg.[3] Although hypertension is necessary to diagnose preeclampsia-eclampsia,[5] it

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From the Department of Maternal-Fetal Medicine, Baptist Hospital, Nashville.

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is not found in the HELLP syndrome at its onset, though most patients will manifest an elevated blood pressure as the disease progresses.

Severe preeclampsia-eclampsia is often misdiagnosed, and on initial examination our patient was thought to have gallbladder dysfunction. Goodlin[2] reported a series of 28 women with severe preeclampsia who were misdiagnosed initially as having cholelithiasis, hepatitis, epilepsy, nephrosis, and other medical conditions. Patients with severe preeclampsia-eclampsia may show gallbladder dysfunction, edema of the gallbladder wall as demonstrated by ultrasound having been reported,[6] and acute liver disease and DIC may result in maternal death. Killam and associates[7] reported a series of five preeclamptic patients with acute liver disease and DIC, all of whom survived, with reversal of their hepatic damage and coagulopathy. One of Weinstein's patients died 50 hours following admission; autopsy revealed ascites, bilateral pleural effusions, central hepatic necrosis, and multiple petechiae of all viscera.[3]

Conservative management of the HELLP syndrome has been suggested. A series of 27 patients fulfilling the criteria for the syndrome were conservatively managed by MacKenna and associates[8] in order to prolong the pregnancy for further fetal maturation. The mean gestational age was 33.5 weeks. Twenty-four of the 27 perinates survived, and there were no maternal deaths, but the present case raises serious concerns regarding such management of patients with the HELLP syndrome; Weinstein[3] in his original article admonishes the clinician that conservative management may be detrimental to survival. Standard treatment for severe preeclampsia-eclampsia, with particular attention to hepatic, renal, and hematologic function, will minimize complications and decrease maternal mortality.

Summary and Conclusions

The HELLP syndrome should be considered in the differential diagnosis of all pregnant women with atypical preeclampsia-eclampsia. Overt hypertension may not be present initially. Laboratory evaluation of hepatic and coagulation functions is essential for diagnosis and management. Maternal well-being should be considered above all else. Conservative therapy of the patient with HELLP syndrome should be abandoned.

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Cocaine-Related Deaths in Memphis and Shelby County

RICHARD C. HARRUFF, M.D., Ph.D. and JERRY T. FRANCISCO, M.D.

Introduction

Illicit cocaine use in the United States has increased markedly in the recent few years,[1] and as indicated by several sources, the same applies for Tennessee. One measure of the magnitude of cocaine use is the number of deaths in which cocaine and/or its metabolites are detected in postmortem blood, urine, or other samples. Such cases, which include all manners of death, have been referred to as "cocaine-related deaths,"[2] regardless of whether or not cocaine was the actual cause of death. In this report, we present our experience with cocaine-related deaths in the Shelby County Medical Examiner's Office over a seven-year period from 1980 through the end of 1986, in order to provide data with which the problem of cocaine abuse in Tennessee may be evaluated and discussed.

Methods and Materials

The toxicology records on computer file at the Shelby County Medical Examiner's Office were searched from 1980 through the end of 1986 for all Medical Examiner cases in which cocaine was detected in the blood, urine, or any other body fluid. The corresponding Medical Examiner records were then reviewed, and for each cocaine-positive case, age, race, sex, and manner and cause of death were tabulated.

Procedures for drug screening and other toxicological analyses were essentially constant over this period, so that differences in numbers of cocaine-related cases represent real phenomena and not just artifactual differences due to improvements in techniques or case selection. Drug screening on urine was performed by both enzyme multiplied immunoassay technique (EMIT), and organic solvent extraction followed by thin-layer chromatography. Blood was screened by gas chromatography of its organic solvent extract. Cocaine identification was confirmed by gas chromatography/mass spectrometry, and cocaine concentrations in samples were quantitated by gas chromatography using an internal standard.

Results

The yearly totals for cocaine-related fatalities in Memphis and Shelby County are shown in Table 1 for each category of manner of death. Accidental deaths included those resulting from traffic accident injuries (nine) and from drug overdose (eight). The other accidental deaths included one each of subdural hemorrhage related to a previous fall, electrocution at a worksite, acute ethanolism, housefire death, and hyperthermia that was partly environmental, with cocaine contributing to the loss of body temperature control.

Accidental deaths from overdose of cocaine alone accounted for six of the eight drug overdose fatalities. In the other two overdose cases, cocaine was present in combination with other drugs. In one, tripelennamine and pentazocine

TABLE 1

YEARLY TOTALS OF COCAINE-RELATED DEATHS BY MANNER OF DEATH

Year	Accident	Homicide	Suicide	Natural	Total
1980	2	0	0	0	2
1981	0	0	1	0	1
1982	0	3	0	1	4
1983	2	1	1	1	5
1984	2	3	1	1	7
1985	3	8	4	0	15
1986	13	30	3	4	50
TOTAL	22	45	10	7	84

From the Office of the Chief Medical Examiner, Shelby County, and State of Tennessee Department of Pathology, University of Tennessee, Memphis.

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were present, and in the other, heroin (morphine) was detected. The blood concentrations in the cocaine-only accidental overdose deaths ranged from 0 to 208 mg/L. The zero concentration was in a 35-year-old white man who also had a fatty liver, a vitreous humor cocaine concentration of 1.1 mg/L, and cocaine metabolites in the blood. The extremely high level of cocaine was found in a 26-year-old white man who passed out with his head in a garbage bag full of cocaine, aspirating a large amount of the material before he died.

Of the ten suicide deaths, eight were by firearms, and the other two were by overdose of drugs. One was by injection of cocaine alone, and the other was by consumption of several drugs, primarily diphenhydramine and methaqualone.

Six of the seven natural deaths were due to atherosclerotic and/or hypertensive cardiovascular disease; subjects ranged in age from 34 to 52 years. The seventh was a 24-year-old white woman who died in status asthmaticus.

Homicides represented the largest category of manner of death (54% of total deaths). All but four of these resulted from firearms; two were killed by stab wounds, and two by beating.

The distribution of cases by age and race is shown in Table 2. In addition to these data, the sex distribution was 73 men and 11 women. Also, of the 45 cases of homicide, 40 were black and 5 were white.

Discussion

These statistics from the Shelby County Medical Examiner's Office demonstrate the dramatic increase in cocaine-related fatalities in the Memphis area over the last six years, and especially within the last two years. This situation is similar to that occurring all over the United States, although the accelerated increase in Tennessee has lagged somewhat in time behind the same type of increase reported in the coastal cities.

All manners of death have shown a several-fold increase, and in each category, cocaine caused or contributed to death in different ways. In most overdose deaths in which only cocaine was present, it was apparent that cocaine directly caused death. For the one case in this category that had a negative blood level, the interpretation of the toxicology findings was less straightforward. In general, because of the postmortem activity of blood esterases, blood concentrations determined after death are often unexpectedly low. For this reason, the vitreous humor concentration more accurately reflects the actual blood level at the time of death, but even this determination is low when death occurs suddenly, before the vitreous humor equilibrates with the blood. Therefore, it is best to recognize that there is no "lethal" level of cocaine; death may occur at any blood concentration, and the determination of cause of death must be based to a large extent on the circumstances surrounding the death.[3]

In deaths due to traffic accident injuries, the contribution of cocaine was not clear. It is likely that cocaine intoxication could encourage risk-taking and interfere with judgment, but in half of the traffic deaths reported here, ethyl alcohol was also present in the blood in levels above 100 mg/dl, thus obscuring any assessment of cocaine's effects.

On the other hand, cocaine's effects certainly may have contributed to the suicide deaths. The severe depression associated with cocaine abuse could have triggered a self-destructive action that otherwise never would have happened. Loss of self-esteem and financial difficulties due to drug use may have been additional factors.[4]

A growing body of evidence indicates that prolonged cocaine use may contribute to or actually cause myocardial ischemia and infarction.[5] Our group of cardiovascular deaths, which were all premature, possibly includes cases of cocaine-induced ischemia, but further studies would be required to allow more than speculation regarding the role of cocaine in them.

Homicides constituted the greatest proportion of cases, as well as the category showing the most rapid increase. Initial evaluation of the cases with this manner of death indicates that quite fre-

TABLE 2

COCAINE-RELATED DEATHS BY AGE AND RACE

Age	Black	White	Total
20-24	6	6	12
25-29	14	8	22
30-34	19	6	25
35-39	6	6	12
40-44	5	3	8
45-49	3	1	4
50-Up	0	1	1
TOTAL	53	31	84

quently dealings or disputes over cocaine precipitated the situation that led to the victim's death. In other instances, the effects of cocaine could explain aggressive and irrational behavior which resulted in lethal actions for self-defense. Overall, this category of deaths provokes major concern, partly because the marked increase in cocaine-related homicides in 1986 coincides with the all-time record number of total homicides per year in Memphis. Moreover, our data, which indicate the drug status of the victims but not the perpetrators of homicide, obviously do not reflect the full contribution of cocaine to violent crime.

Regarding the age distribution of cocaine-related deaths, most of the deaths were in the age range of 25 to 34 years, an age distribution that may change in the future as usage of the drug becomes more prevalent in the younger age groups. Also, it is possible that there will be an increased incidence of cardiovascular deaths caused by prolonged cocaine abuse in younger populations, but it is likely that few of these will be recognized as such.

The overall racial distribution of the deaths is predominantly black, with a black/white ratio of 53/31, although if the homicides are subtracted, this ratio becomes 13/26 for non-homicide deaths, indicating that blacks using cocaine are more likely to be murdered, while whites involved with the drug may die as a result of an accident, which may be a drug overdose, as a result of suicide, or from an apparently natural "heart attack" promoted by cocaine abuse.

In conclusion, there is no doubt that cocaine abuse has quickly become a major public health and social problem. Cocaine is by no means a safe drug, and it does cause and contribute to death by a number of mechanisms. It causes overdose deaths that are unpredictable with respect to dosage, it leads to depression that may precipitate suicide, and it may increase the possibility of a fatal accident. Most alarming is the drug's contribution to violent behavior. Continuing and future analyses of cocaine-related homicides recorded in this office will provide a more accurate estimation of the extent to which cocaine contributes to homicide and violent crime, and of the mechanisms by which it does so. In the meantime, public awareness of these and similar data should aid in formulation of specific policies and actions needed to counteract the growing problem and to limit the future numbers of cocaine-related deaths.

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Special Item

Vanderbilt Medical Internship and Residency—Past and Present

LAURENCE A. GROSSMAN, M.D.

More than 45 years have passed since completion of my medical internship at Vanderbilt University Hospital. The contrast between 1941, its date, and the present program reflects changes in medicine, its evolution in society, the aging population, and the expectations of the young physician.

Organization

Vanderbilt's medical internship and residency programs began in 1925 and were patterned almost identically after the residency training program of Dr. Warren Longcope's medical service at the Johns Hopkins Hospital. Prior to this, the initial impetus and concept came from Dr. William Osler, Dr. Longcope's immediate predecessor.

There was little if any subspecialty training; hematology, oncology, infectious disease, or cardiology divisions did not exist. The therapeutic armamentarium was limited to neoarsphenamine, bismuth, quinine, phenobarbitol, insulin, atropine, morphine, codeine, Lugol's solution, and salicylates. There were no antimicrobials. Mechanical therapy for infectious diseases was employed. A hot box was used for gonococcal arthritis, and other forms of non-genital gonococcal disease. Pneumothorax and pneumoperitoneum were used for treating tuberculosis. The only subspecialty clinics were the allergy clinic, the "L" (lues) clinic, and the chest clinic. By far the most popular and best attended of them all was "L" clinic, three mornings and one evening each week, the night clinic often lasting until 11:00 PM.

Disease Spectrum

The disease spectrum was totally different from today's. Usually, one-fourth to one-third of all patients had bacterial pneumonia; each proved to be a challenge to the house officer, particularly the intern. The leukocyte count gave some indication as to prognosis—poor when counts were high, worse when low. The only treatment available was type-specific antiserum. The selection of the appropriate antiserum for pneumonia due to pneumococci necessitated a flurry of activity. The throat was swabbed, and sputum was obtained in an endeavor to see if pneumococci were present, and if they were, whether they could be typed directly. Blood cultures were drawn. Sputum and throat washings were injected immediately into the mouse's peritoneal cavity, a semiselective culture medium for pneumococci requiring only eight hours for maximum growth. After typing the pneumococcus and finding the type of specific antiserum, the patient was tested for serum allergy, and if it was negative, antiserum was administered—always under close observation. This sequence of events required eight to ten hours of the intern's time! Despite such efforts, a mortality of 20% to 25% was usual.

Though tuberculosis was rampant, in retrospect it was what was termed tuberculosis that was rampant; there was no knowledge of histoplasmosis, avian mycobacterial infections, and the like. On one occasion, 23 of the 28 patients on the men's medical floor were diagnosed as having some form of tuberculosis—pulmonary, bone, joint, meningeal, etc. The disease extended to the housestaff and the student body. No class, including my own, escaped without infection.

Syphilis was also present in all forms—primary, secondary, and tertiary disease, including tabes dorsalis, paresis, gumma, aneurysms, etc.

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Reprint requests to St. Thomas Medical Group, 4230 Harding Road, Suite 400, Nashville, TN 37205 (Dr. Grossman).

Throughout the entire period of housestaff training, there was only one patient with lupus, and he was exhibited many times on various attending rounds. Myocardial infarction was uncommon, but peptic ulcers, often bleeding, were frequent. Cancer of the lung and cancer of the stomach were rare. Glomerulonephritis was prevalent.

Housestaff

The medical housestaff consisted of five interns, four assistant residents, and a chief resident—a total of ten (Fig. 1). In addition, ten rotating internships were offered at the Nashville General Hospital under a separate program. When considering the housestaff size, one must recall that there was no Veterans Hospital, and no St. Thomas Hospital housestaff. All members of the housestaff lived in the hospital. Interns' compensation consisted of room, board, uniforms and laundry, and for assistant residents that was increased $25 per month.

In 1946 I was an assistant resident after having spent four years as an army medical officer. There were four classes returning after periods of military service. No housestaff positions were available. Many worked in the medical outpatient department, and simply attended rounds. For this they received credit for one year of training. The medical residency program was devised as a pyramid, with survival of the fittest aptly describing the system. After a year of assistant residency, one was expected to spend the following year in pathology. Only one man progressed to the chief residency. Other individuals sought additional training and began to drift toward the developing subspecialties. Vanderbilt had a special relationship with Johns Hopkins Hospital. There were frequent brief or sometimes longer rotations of assistant residents between the two institutions. Many of the department heads at Vanderbilt were Johns Hopkins graduates.

The program was grueling. You were on call 24 hours each day, and it was necessary to sign

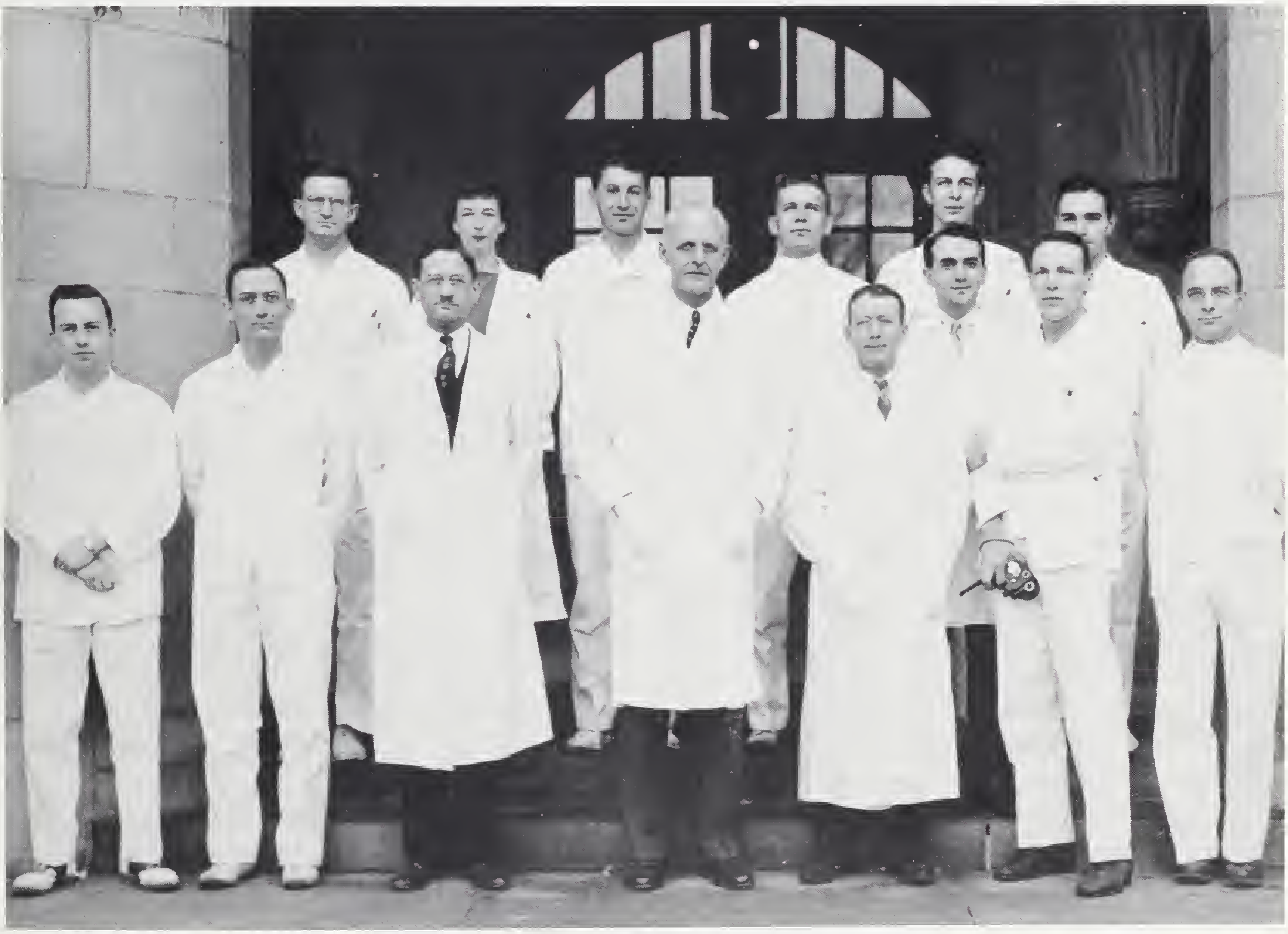

Figure 1. (Front row, left to right) Drs. Malcolm J. Mann, Benjamin P. Folk, Jr., Rudolph H. Kampmeier, Hugh Morgan, John Youmans, A. McGehee Harvey, Frederick T. (Josh) Billings, Theodore B. (Pete) Steinhausen; (back row, left to right) Drs. Thomas A. Donnell, Dorothy M. Horstman, Laurence A. Grossman, Blair V. (Val) Jager, Joe H. Crampton, Robert D. Beech.

out to another house officer if you left. Promptly on your return, your responsibilities resumed. Of the 28 beds on each ward, 26 were for staff patients and under the direct responsibility of the assistant resident physician. No malpractice or legal problems, no social service support, and no financial problems existed. The hospital was supported by its endowment. Nurses were poorly paid, and interns were not paid at all. There were few, if any, drugs, and little support equipment was required or available. The hospital personnel were on the lowest financial pay level. Nevertheless, their work was superb.

Marriage for the intern was out of the question. One of our group was married, but he had been prior to enrolling in medical school. In midyear an unusual event occurred; the announcement of the engagement and coming marriage of one of us appeared in the *Nashville Banner.* Dr. Hugh Morgan, chairman of the Department of Medicine, called the young doctor into his office and inquired about the news item. The intern explained to Dr. Morgan that he attempted to consult him prior to the announcement, but that Dr. Morgan had been out of town, possibly related to his imminent assignment as chief of medicine for the United States Army Medical Corps during World War II. After the explanation, Dr. Morgan informed him that he could leave the day after he was married, and that he entertained serious doubts that another worthwhile medical internship program would be available anywhere in the entire country. Needless to say, the marriage did not take place.

Internship duties involved total care of the patient. There were no intensive care units, no hematological laboratory. If a blood sugar determination was needed at night, the intern went to the blood chemistry laboratory and carried out the test. Sulfanilamide made its appearance in 1941; that meant that a daily white count, hemoglobin, differential count and urinalysis were required, and you did them unless the students were present. Then, as now, diagnosis was challenging; knowledge was limited, and medical instruments primitive. I recall carrying out a consultation in the emergency room—a young woman with severe abdominal pain, and the surgical resident was in doubt as to the diagnosis, but believed that surgery was not indicated. The gynecology resident had examined the patient, and ruled out any pelvic pathology. Finally, as the medical resident on call, I noted that the urine specimen, which had been placed on the window sill, had turned a dark burgundy in color. Suspecting that she had hematuria and possible renal disease, I was surprised to find no red cells on microscopic study, and that the urine did not contain any hemoglobin. Taking the specimen to biochemistry and using a Wood spectrascope, it became evident that the color was due to porphyrins. What a way to make such a diagnosis! Actually, porphyria had recently been described, but we did not know it at the time.

Procedures and tests were primitive. There were only three leads on an electrocardiographic study. There was one central machine, and wires extended to each ward. It was only during my residency that lead CF 4 was added. Peritoneal dialysis had just been described. We had a patient who had been anuric for nine days after a transfusion. Osmolality was poorly described, and really not known. There were no bottled fluids. We spent all night determining the electrolyte composition of the peritoneal dialysate, and preparing and sterilizing the fluids, using five gallon vats. Later a cannula was placed in both sides of the peritoneal cavity, one tube being used for fluid infusion, the other for fluid withdrawal. The patient survived and became entirely well, and the case was duly reported.[1]

Early Vanderbilt History

Full-time faculty totaled four: Dr. Hugh Morgan, chairman of our department, Dr. Rudolph Kampmeier, Dr. Edgar Jones, and Dr. A. McGehee Harvey. There were five others of the clinical faculty who shared the brunt of teaching housestaff and students: Dr. Clarence Thomas, Dr. Albert Weinstein, Dr. David Strayhorn, Dr. Hollis Johnson, and Dr. Sam Riven. Dr. Tinsley Harrison and Dr. John Youmans had just departed, and their absence was notable. Physical findings were stressed, demonstrated at length, and minutely documented. Very few other diagnostic aids were available. Dr. Morgan was the master physician. His examination notes were thorough and detailed every physical finding, commenting both on the patient's personality and his response to his illness. His Christ-like appearance was accompanied by an empathy never equaled in my experience. While not an outstanding speaker, his lectures were nevertheless superb in content. No one ever made physical diagnosis more attractive and more explicit than Dr. Rudy Kampmeier. He was admired by all the housestaff and student body. Dr. Edgar (Bunny) Jones was always available for hematology advice, and clinical pathological consultation. Dr. McGehee Harvey seemed interested in neurological research, and spent little, if any, time with students or residents.

The high point of each week was the clinical pathological conference. The pathologist was either Dr. Ernest Goodpasture or Dr. James ("Lean Dog") Dawson, and each managed to find the most bizarre and unusual cases—scenarios that make the current presentations in the *New England Journal of Medicine* seem simple. Dr. Tinsley Harrison always gave an animated discussion. His monkey-like pacing in the front of the amphitheater is still imprinted on my memory. On one occasion Dr. Harrison and Dr. William Dock jointly conducted the CPC. Dr. Dock had just returned from debating with Harry Bridges in San Francisco. Mr. Bridges was at that time head of the longshoreman's union. No theater offered better drama or more entertainment than this program. It is strange how the passage of time alters one's concept of a physician. As a student, I saw Dr. Harrison as having all the right answers; each step was physiologically explained and dynamically expressed. In contrast, Dr. Morgan's approach seemed less impressive, even rather dull at times. Later, returning after a four-year hiatus of military service, a review of the patients' charts on subsequent admission was indeed astounding. Dr. Morgan was invariably accurate, and without fault or error. Often Dr. Harrison's spectacular diagnosis evaporated with time. What a contrast!

Changes Following World War II

World War II changed the composition of the training program. Returning physicians were older; they were married, and had children. With the completion of the Veterans Administration facilities, the number of available positions more than doubled; subspecialty programs officially came into existence. The emphasis on the pathology rotation subsided. The pyramid system leading to a chief residency was modified, and the importance of the position downgraded. Most investigative work had been carried out by clinicians; now, following Dr. Flexner's report and recommendation, a new breed of doctor, primarily interested in research and experimental medicine, appeared. Fellowship programs developed as medicine became more complex, and subspecialization attracted a greater number of residents. With the advent of Medicare, various forms of government funding, HMOs, PPOs, and other health care patterns of reimbursement, general internal medicine is today becoming less attractive to the physician in his residency training, and fewer physicians elect this broad specialty.

Present Vanderbilt Training

Today's intern earns at least $15,000 per year, and the salaries of residents and fellows range from $20,000 to $30,000 per annum. House officers are often married, and have families. Though time commitments continue to be great, they have lessened. Interns and residents are bright, hardworking, and more attractive; each year they are better rounded and educated. To most, medicine still offers the enchantment, the excitement, the thrill of the unknown, and the reward of patient appreciation.

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Isolated Blunt Disruption of the Common Bile Duct

EUGENE C. MANGIANTE, M.D.; LYTLE BROWN, M.D.; and
TIMOTHY C. FABIAN, M.D.

Introduction

Disruption of the extrahepatic biliary tree by blunt trauma is usually associated with massive, complex, and often fatal injury to the right upper quadrant. This uncommon entity is most frequently detected during abdominal exploration after control of injuries requiring more urgent attention. Such isolated injury almost uniformly escapes early diagnosis and remains masked until obstructive jaundice follows stricture formation. The following case exemplifies the meager early findings, and outlines a rational plan for surgical repair.

Case Report

A 38-year-old white woman entered the emergency room with abdominal pain and distension 30 hours after being involved in a motor vehicle accident. The blood pressure was 140/80 mm Hg, pulse rate 120/min, and temperature 38.2°C, and there was diffuse abdominal tenderness and guarding, without bowel sounds. The hematocrit was 38%, WBC count 10,600/cu mm, and serum and urine amylase 254 and 1,240 units, respectively. The patient was taken to the operating room with a diagnosis of pancreatic injury. At operation, 500 ml of blood-tinged and bilious fluid was noted in the abdominal cavity with bile staining in the paraduodenal area. A 2-cm longitudinal tear was noted in the lateral, distal portion of a normal sized common bile duct. The remainder of the intra-abdominal and retroperitoneal structures were normal. Primary repair was performed with 6-0 interrupted polypropylene sutures and a T-tube placed through a separate proximal choledochotomy. The patient was discharged 12 days after surgery; she inadvertently dislodged her T-tube one week later, and six months after injury she was asymptomatic.

Discussion

A total of 94 cases of isolated extrahepatic biliary ductal injuries have been reported.[1-7] In recent years they are almost uniformly the result of motor vehicle accidents. Although still very uncommon, blunt ductal injury occurs more frequently in association with injury to the pancreas, duodenum, liver, and adjacent vasculature. The distal common bile duct at the superior pancreatic border is the most frequent site of blunt injury, followed by the common hepatic duct. The degree of injury ranges from complete transection to contusion with resultant ischemic stricture formation.

Several theories of ductal injury have been proposed. Compression between the abdominal wall and the vertebral column,[8] although possible, would seem unlikely to injure only the bile duct. It has been suggested that a sudden increase in intraluminal pressure from gallbladder compression might lead to ductal rupture.[9] One quite plausible theory suggests the application of blunt force to the abdominal wall with sudden, rapid superior displacement of the liver.[10] Additionally, traction on the biliary tree as a result of hepatic displacement may explain why the most common site of injury is at the superior border of the pancreas, a point where the duct is relatively fixed.[11]

Isolated injury may cause only minimal early abdominal symptomatology because of the initial retroperitoneal containment of sterile bile. Even with intraperitoneal bile, a peritoneal response may still not be elicited without concurrent bacterial contamination. Unless physical examination, peritoneal lavage, or computerized tomography disclose concurrent intra-abdominal injuries, the diagnosis of bile duct injury is seldom initially suspected. Several reports[3,8,12,13] have described an early clinical pattern following blunt abdominal trauma in isolated injury of the bile duct. There may be initial mild hypotension.

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Peritoneal lavage will not reveal hemoperitoneum without associated injuries, and essentially all symptomatology resolves over the next 24 hours. The ensuing one to ten days are characterized by abdominal discomfort and/or distension, anorexia, nausea, vomiting, or jaundice. Hyperbilirubinemia and leukocytosis may develop, and the triad of jaundice, biliary ascites, and a recent history of blunt abdominal trauma is diagnostic of injury of the bile duct.

Surgical management is dependent upon both the location and extent of injury. If there is not a segmental loss of extrahepatic duct, or retraction of the duct distal to the injury into the pancreas, the lesion is often amenable to simple repair.[2] A monofilament synthetic suture may be used, and interrupted sutures appear superior in the prevention of luminal compromise. Most authors have advocated placement of a choledochostomy tube through a separate choledochotomy.[12]

Injuries not conducive to primary repair can be treated by either Roux-en-Y choledochojejunostomy or choledochoduodenostomy. Those cases managed by primary repair or choledochoenteric anastomosis should generally have a stent because of the diameter of the common duct. Attempts to identify a duct which has retracted into the pancreas, or a duodenotomy with retrograde identification, are unwarranted and often convert a single simple injury into a more complex one.[14] Closed external drainage is adequate for the retracted distal duct. Cholecystojejunostomy can be performed rapidly and should be considered in patients with other serious injuries. If this fails, a significant amount of native duct is available for secondary reconstruction. Long-term follow-up is important in the management of the patients. Stricture formation will be heralded by jaundice and abnormal liver function tests.[2,4] Although most strictures clinically manifest themselves early, some do not occur for years; therefore, these patients should be followed for life.

The occurrence of a blunt disruption of the extrahepatic biliary tree is not only rare, but the preoperative diagnosis is seldom made in the acute phase. The isolated ductal injury usually manifests itself as obstructive jaundice due to stricture formation days to weeks after the initial injury. Repair must be based upon the location and the extent of injury. Long-term follow-up is essential due to the frequency of postoperative strictures.

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Indoor Radon

JACKIE WAYNICK

With all the news stories about radon, it is likely that physicians will be receiving questions about it. Radon is colorless, odorless, and tasteless radioactive gas produced in the decay of uranium, which is present in varying amounts in soil and rocks throughout the country. Since it is readily diluted in outdoor air, radon is dangerous only when it becomes trapped in confined places, such as inside buildings. In recent years, scientists have discovered that if the soil under a building contains decaying uranium, radon can accumulate in the structure in amounts depending upon the uranium content and porosity of the soil, the type of construction of the building, and meteorological conditions.

Radon is an inert gas with a half-life of 3.8 days, but when it decays it produces a series of short-lived (half-life less than 30 minutes), radioactive products called radon progeny or daughters. If inhaled, these radon daughters may become trapped in the lungs where they undergo further decay and can damage lung tissue.

The only known health effect associated with exposure to radon is an increased risk of lung cancer; it has been estimated that 5,000 to 20,000 lung cancer deaths each year in the United States may be attributed to radon,[1] which would represent 4% to 15% of the total lung cancer deaths each year. In addition, scientific evidence indicates that smoking can increase the risk.

Radon enters buildings by percolating through the soil and rock and then seeping through dirt floors, cracks in concrete floors and walls, floor drains, sumps, joints, and tiny cracks or pores in hollow-block walls.[2] Radon can also enter water within private wells and be released into the air inside a building when the water is used,[2] though this is not believed to be a problem in Tennessee.

In order to determine the level of radon in a building, the air must be tested with proper equipment. The two most popular commercially available home radon detectors are the charcoal canister and the alpha track detector, both of which are exposed to air in the home for a specified period and then sent to a laboratory for analysis. The charcoal canister can be purchased for $12 to $25 and requires an exposure of three to seven days; the alpha track detector costs from $20 to $50 and can be used to collect a sample over a period of two weeks to several months. Other techniques for measuring radon are more expensive and require trained personnel. Results from devices that measure radon decay products are reported as "working levels" (WL), and results from devices that measure concentrations of radon gas are reported as "picocuries per liter" (pCi/L).

The Tennessee Division of Air Pollution Control of the Department of Health and Environment can provide homeowners interested in making radon measurements with a list of laboratories and businesses who have demonstrated their capabilities in measuring indoor radon to the Environmental Protection Agency.

To evaluate a home for radon, one generally starts by making a short-term screening measurement to get an idea of the highest level in the home. This measurement should be made in the lowest living area of the home. All windows and doors should be closed for at least 12 hours prior to the start of the test and kept closed as much as possible throughout the testing period to keep the radon level relatively constant.

Generally, the screening measurement alone will not provide a reliable average, but rather serves to indicate the potential for a radon problem. If the screening measurement is less than 4.0 pCi/L additional testing is probably not indicated. However, if a screening measurement is

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From the Division of Air Pollution Control, Tennessee Department of Health and Environment, Nashville.

greater than 4.0 pCi/L it is advisable to follow-up with a detection device designed to sample the air over a longer period, which should provide an idea of the average concentration throughout the house.

An individual's actual risk depends upon the time of exposure to a particular concentration. One way to think about the risk associated with radon exposure is to compare it with the risk from other activities. For instance, if an individual spends 75% of each day throughout his lifetime in a dwelling which had an average radon concentration of 3.0 pCi/L, his risk of developing lung cancer would be comparable to the risk of receiving 200 chest x-rays per year. The same length of exposure to an average concentration of 15.0 pCi/L would be comparable to the risk of smoking one pack of cigarettes a day. Other factors, in addition to smoking, may further increase the risk.

Some actions that one might take to immediately reduce the risk from radon include stopping smoking in the home, spending less time in areas with the higher concentrations of radon, and opening windows and using fans whenever practical to provide flow-through ventilation. If the house has a crawl space, the crawl-space vents should be kept open all year.[1]

While these actions will help reduce an individual's risk from radon, they do not offer a long-term solution. More information about permanent measures to solve radon problems and those caused by other common indoor air pollutants such as formaldehyde, pesticides, asbestos, and biologicals may be obtained from the Tennessee Division of Air Pollution Control, 4th Floor, Old Customs House, 701 Broadway, Nashville, TN 37219-5403, telephone (615) 741-3931.

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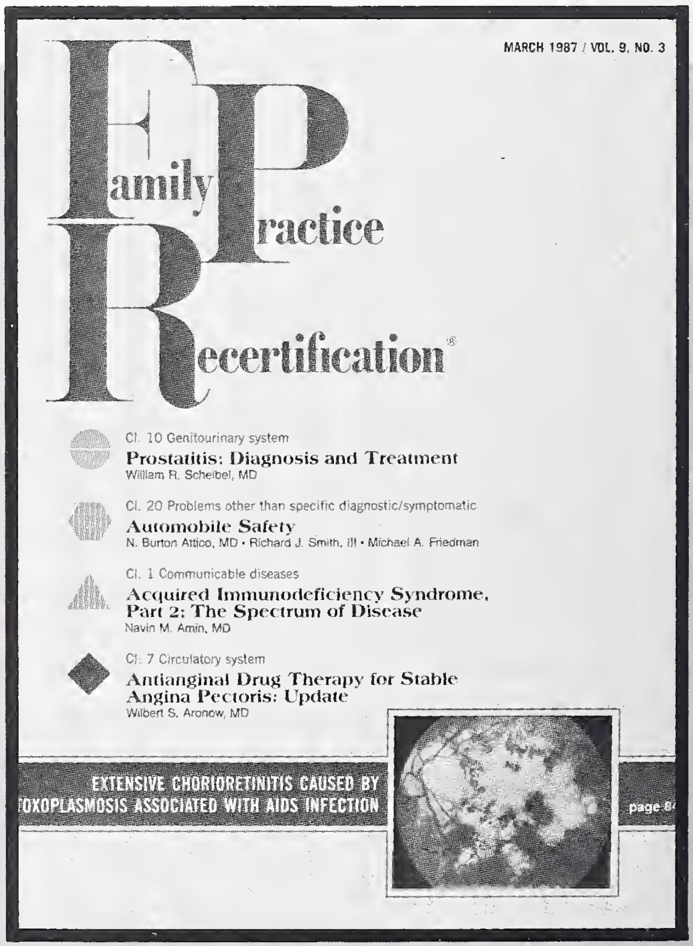

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Too Good To Be True!

J. KELLEY AVERY, M.D.

Case Report

A 67-year-old woman was admitted to a community hospital from her physician's office with a history of generalized abdominal pain for 24 hours and nausea with vomiting for 12-hour duration; the patient denied any prior trauma or unusual dietary intake, though she did give a history of two previous episodes of transient upper abdominal pain during the past month, occurring within 30 minutes after a meal and accompanied by some belching and a sensation of fullness. The pain was nonradiating and subsided spontaneously after 30 to 45 minutes.

The present illness began 15 to 20 minutes after a normal meal, and persisted, becoming gradually more intense. There had been some colicy symptoms following onset and four loose, watery stools during the first eight hours of the present attack. The patient stated, "My stomach feels like it will burst."

Past history was unremarkable; she reported no previous surgery.

The patient's attending physician was a board-eligible general surgeon who had seen her in his office with the second of her transient attacks, but at that time the pain was subsiding and the examination revealed no reason to further evaluate the patient.

The physical examination done at the visit prior to hospitalization revealed a temperature of 99°F, blood pressure 160/70 mm Hg, pulse 88/min, respiratory rate 14/min. The positive findings were limited to the abdomen, which was generally tender with minimal splinting but no rebound tenderness or localization. Bowel sounds were present and questionably hypoactive, and though there was possibly some distension, the patient was moderately obese and this was difficult to evaluate.

After admission the WBC count was 9,000/cu mm with normal differential. Blood chemistries were within normal limits including glucose, creatinine, and electrolytes. KUB showed some large and small bowel gas but no definite distension, and the radiologist who reported on the film the morning after admission gave "ileus" as his impression. The patient was ordered NPO and intravenous fluids (D 5-NS) at 125 cc/hr were begun.

The morning after admission, Saturday, the surgeon made rounds and found the patient about the same, having passed scant flatus, but no stools. Her abdomen was essentially unchanged but did appear more distended. An NG tube was passed and about 250 cc of bile-stained, guaiac-negative, material was obtained. IV fluids were continued and a repeat KUB, CBC, urinalysis, and electrolytes were ordered for the morning. The patient was not seen by her attending physician the rest of that day, during which her pain gradually became worse, some vomiting occurred around the NG tube, and the temperature rose to 101°F.

The attending physician found his patient on Sunday morning feeling worse, having more pain and definitely more distended. The KUB was read by the attending physician as "essentially unchanged" from the admission file; the radiologist was not called. The WBC count was 11,000/cu mm with 20% bands forms. The serum sodium was 130 mEq/L, but the potassium was 2.8 mEq/L. The abdomen was more tender, and there was some rebound tenderness and guarding in the right lower quadrant; bowel sounds were not heard.

The patient was taken to surgery with the suspicion of a ruptured appendix. On entering the abdomen the surgeon found a large amount of serosanguinous fluid with a foul odor, and exploration revealed a massive infection of the small bowel with multiple perforations. Resection and anastomosis was accomplished, but, despite massive antibiotic therapy and aggressive support, the patient died on the third postoperative day.

A lawsuit was filed charging negligent delay of diagnosis as the cause of death.

Loss Prevention Comments

As the case developed, the defendant physician had several problems. He saw his patient only once during the first 36 hours of her hospital stay. On careful reevaluation of the x-rays, some calcification could be seen in the upper abdominal aorta, which might have alerted him to the possibility of ischemic bowel disease, but despite the seriousness of the situation, expert testimony was available to demonstrate that the physician performed within an acceptable standard of care, and that, even with the most aggressive efforts of diagnosis and treatment, chances of recovery were almost nonexistent.

The defendant did well with this deposition despite a vigorous and lengthy examination by the plaintiff attorney, and the case looked extremely good for him. The plaintiff attorney became suspicious that things might not be as they appeared, however, and thought that the office records were "too good to be true." Under oath, the defendant's nurse testified that about ten days after the lawsuit was filed, she was asked to bring the patient's office records into the physician's

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Dr. Avery is chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood, and medical director for Ambulatory/Outpatient Services, St. Thomas Hospital, Nashville.

office and together they altered the records by rewriting each entry; they then destroyed the original medical record. Although the defendant testified that the alterations were made to more clearly indicate what really happened, his credibility was lost, a large settlement was paid and the surgeon's professional liability was not renewed.

MORAL: *Don't Alter Files After a Lawsuit Has Been Filed, Even To Correct an Obvious Error!* It is much easier to defend mistakes than to try to restore credibility.

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2100 West End Avenue, Suite 725 - Nashville, Tennessee 37203 - (615) 329-4770

JAMES T. GALYON

Catastrophic Health Coverage

Your TMA recently sent a group of physicians to Washington to meet with our congressional delegation. While there we learned that some catastrophic health care bill would almost certainly be passed by Congress this year. The major uncertainties appear to be in the final form of the bill that will be passed.

It is hard for us as a group of compassionate, caring physicians to be against a proposal to protect the elderly from an economic disaster—the loss of all of their resources late in life without the realistic opportunity to recover from this blow. We do view this legislative activity with some degree of caution and even distrust. Catastrophic coverage has long been feared as the nose of the camel in the tent. This could readily be expanded to a national health insurance plan by lowering the threshold of the entrance into the program or, by allowing inflation to do this for Congress, by maintaining the same threshold year after year in the face of an annual inflation.

The current legislation appears to pose only some of these threats. The legislation expected to pass this year would apply only to Medicare beneficiaries, initially. This new insurance would be voluntary and would require additional monthly premiums of approximately $6, or $72 a year. There would be a cap on the out-of-pocket Medicare covered expenses, and this could range from $1,700 to $2,000 annually. This bill is supposed to be budget-negative, i.e., neither costing the federal budget anything nor raising any new revenue. There may be taxes on the Medicare benefits received by some, based on a formula related to income.

Other amendments or proposals are being mentioned in Washington. These include extending the coverage to other than Medicare beneficiaries and to include drug expenses in this bill. The cost of this latter proposal is unknown, but it is expected to have a major fiscal impact on the bill.

Dr. Otis Bowen, Secretary of the Department of Health and Human Services, has long been a proponent of this type of legislation, and now the President is backing this bill if it can be made budget-negative.

Our best role in this activity is to carefully watch the legislative procedure and to actively advise Congress and the administration. We want this bill limited to those already insured by the federal government, i.e., Medicare and Medicaid recipients. There is little need for the government to get further into the insurance business. It is my impression our lobbyists in Washington are carrying out just this oversight activity.

James T. Galyon, M.D.

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tennessee
medical association

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AUGUST, 1987

editorials

Med Ed— Yorn, Mine, His'n, an' Her'n

The degradation of the once almighty Yankee dollar is nowhere more apparent than in the cost of a medical education. Perhaps the $400 tuition my father paid to send me to Vanderbilt was as costly to him in terms of real dollars as today's tuition is to its victims, but it long since passed a factor of 10—in fact, when my son matriculated there nearly 20 years ago—and it has nearly tripled since that time. The dollar has fallen in grace, to be sure, but not that much.

In addition, while I was at Vanderbilt, tuition at the University of Tennessee Medical Units in Memphis was negligible, most of the cost being borne by the taxpayers. Not anymore. Tuition at tax-supported institutions today is substantial, even for state residents. For non-residents it is often comparable to that of privately funded schools. Why the change?

Inflation has hit the medical schools as it has everything else, but the increases, particularly in the last few years, have been vastly disproportionate, despite substantial subsidization of the students' education by the institutions. It is just that the same factors affecting health care generally have been having their impact on medical education; foremost among them is increasing sophistication of procedures and techniques that simply cost more to do for the patient and demonstrate to the student. Technologic advances have also spilled over into the classroom and the teaching process as well. Not insignificant either is the cost of just attending to the ponderous bureaucratically generated paperwork, which consumes a progressively greater proportion of the institution's time, energy, and funds.

The other day I received a letter of thanks from the Dean of Vanderbilt's School of Medicine for support during the past year, in which he detailed some of the institution's achievements over the past year, including the addition in the past five years of 120 new faculty members, recruitment of 11 new department chairmen (including some of new departments), and initiation of 50 new programs. He pointed out that he views this spending as an investment in the future rather than money spent never to be seen again. So do I.

My late friend and mentor, John L. Shapiro, M.D., once told me that he could not imagine a doctor's not making medical education his major charity. I know for a fact that he gave of himself unstintingly to that end, and I feel certain that he did the same with his worldly goods. The presence at the recent annual dinner of the Canby Robinson Society, Vanderbilt Medical School's major support group, of so many of his former students and colleagues, among them many members of the Vanderbilt faculty, attested to the success with which he and others of like mind passed along their enthusiasm for this philanthropic aspect of medical education.

The philosophy of the current federal administration is to involve the federal government in medical education as little as possible. I have no quarrel with that, as I am a states rights man myself, and believe that to be a responsibility of the states. Nevertheless, the whole cost of a superior medical education these days is a burden too great for even the combined tuition and tax funds. The needs of our public institutions are not dissimilar to those of the privately funded ones, since each has vast requirements over and above its basic sources.

An individual's educational institution is not called *alma mater* for nothing; it is our cherished mother, the place where we were nurtured in, among others, things medical. As with one's natural mother, she has a lifetime claim upon our resources commensurate with her needs and our abilities. However much we may attend to those claims, considering the debt we owe, they are never met.

In the oath that bears his name, Hippocrates addressed the responsibility of the student toward his teachers. It is easy to say that, Oh, well, in his day the teacher *was* the school; things are different now. The school is just a mass of bricks and mortar. Wrong. The mass of bricks and mortar is just where the school hangs its collective hat. The school is people. Our obligation did not cease when we received our degree of Doctor of Medicine. In fact, it just began.

Your gift to your medical school is a lot more than the fulfillment of an obligation, however. Your practice remains strong only so long as medicine remains strong, and medicine's strength is tied irrevocably and inextricably to the health of our medical schools. Your gift, as Dean John Chapman opined, is also an investment in the future. But the future you are investing in is not just your medical school's, or even medicine's. It is also your own.

Though quoted out of context, to be sure, a new context I give unto you: "A word to the wise is sufficient." "Go Thou and do likewise." "A penny saved is a penny earned." "The life you save could be your own."

J.B.T.

On Epidemics

Back in the days before it was considered appropriate, or even proper, for women to be noticed outside the context of the family, women found ways of circumventing convention and insinuating themselves into the world of men. In his play *Lysistrata*, Aristophanes made it clear some millenia ago that women have always held the upper hand in running the world, male supremacy being only in the eyes of the male beholder, but I am speaking of the outside world. One of those ways was simply to assume, for whatever purpose, a masculine name. One author that comes to mind is George Eliot. There were others.

To while away her lonely, often tormented days on her farm in Africa, the Baroness Blixen wrote her immensely popular *Seven Gothic Tales*, published in 1934 under the pen name Isak Dinesen. When at the spinsterish age of 28 Karen Dinesen married her cousin Bror Blixen, he gave her two wedding gifts: he made her a baroness, and he took her to Africa. He also gave her syphilis. Her marvelous autobiographical *Out of Africa*, the account of her Africa years—1914 to 1931—smoothes over the anguish of her broken marriage and the torments of the *tabes dorsalis* she acquired and lived with until she died in 1962 at the age of 77.

Whether syphilis was a present of the Indians to the European invaders of this hemisphere, or vice versa, is unclear, there being evidence to support either view. With typical Western chauvinism the former has until recently been assumed, but perhaps incorrectly. In any case, the conquest of the new world and the conquest of the Conquistadores coincided, and syphilis became the bane of Europe in the 16th and 17th centuries, being known to the English as the "French disease," to the French as the "Italian disease," and to the Italians as the "Spanish disease." Though it quieted down some after that, whether due to attenuation of the microorganism or to adaptation by the human organism, or both, it is still with us. It is still with us despite penicillin, to which the organism is extremely sensitive, and despite the efforts of Surgeon-General Thomas Parran to bring it out of the backrooms and the gutter into the clinics and drawing rooms with his ultra-shocking book *Why Don't We Stamp Out Syphilis?*, in which *that word* actually appeared in respectable non-medical bookstores for all to see. The year was 1938, the year I was graduated from high school.

It is hard to hide syphilis from its victim. Early in the game a chancre shows up somewhere (though in women and male homosexuals it may be out of sight). Soon it becomes hard to hide from anyone unless the patient can be convincing as a case of measles. Dr. Parran set in motion a massive case-finding effort in which contacts were sought out and treated; serologic studies were done on every hospital admission and were required for marriage licenses. Though treatment at that time was tedious and uncomfortable, arsenic and bismuth did work most of the time, and it was only a few years until penicillin seemed on the verge of actually accomplishing what Dr. Parran had initiated.

There are similarities between the plague of syphilis as it occurred in 16th century Europe, and our present sexually transmitted plague, but there are even greater differences. Skin manifestations were universal and severe, so that syphilis could not be hidden. Human immunodeficiency virus (HIV) infection is only serologically apparent, sometimes for years and perhaps even sometimes forever. Syphilis was transmissible by any sort of contact, whereas HIV infection is limited to sexual contact and inoculation with infected blood. The differences are even greater between HIV infection and syphilis as it exists today. Syphilis is now generally a minor disease except as it might affect offspring, or in case it should go undiscovered; in such a situation even, most people at some time will receive serendipitously a course of antibiotic therapy to which the organism is sensitive, and be cured. We simply do not see the late manifestations of syphilis anymore.

HIV, on the other hand, is something else again. In the first place, should it eventuate in the acquired immune deficiency syndrome (AIDS), there is no treatment save supportive, and thus far, certain death after a sometimes prolonged debilitating illness. Until AIDS, or at least the AIDS related complex (ARC) occurs, infection is undetectable except serologically, and it is possible for the disease to be spread around willy-nilly. This is the argument for mandatory testing in the way the population was serologically tested for syphilis. There is, however, one predominant difference, and that one difference is staggering. In the one case you uncover a curable disease, and in the other a likely death sentence.

The ethical, moral, and legal morass that all this has dropped us into is stupifying, and new implications are continually appearing. What of the individual who knows he has AIDS and decides to take others with him? Just to imagine that such a situation could possibly exist at all is repugnant, and yet it has already arisen. And what of the young sexually promiscuous boy who has been found HIV positive? How does society protect itself from such profligacy? How do health care personnel—you and I and our employees—protect ourselves? Innocent victims are increasing in number by the day, and due to proliferate even more.

The legal implications are complex, as well. One might find himself in legal jeopardy by either divulging or not divulging confidential information—from the patient if he did, or from the victim should the patient pass his virus along. Is a physician's first duty to his patient or to the public? And where does the duty of the public health official lie? The situation is comparable to that faced by psychiatrists who loose a once psychotic killer on society as cured. One can never know when any victim's anger at his plight or the neuropsychologic consequences of his disease will require vengeance, and turn him on society.

Though much has been said about the loss to society of so many of its most talented members through AIDS, there is an even greater loss to society generally in terms of effort and resources. There is a progressively greater preoccupation not only with the disease itself, but with consequences such as those I have described. There is no member of society that is not in some way affected at least indirectly, if only by the sister syndrome of AFRAIDS, which, if it makes us callous, insensitive, fearful, and suspicious will in the long run do humanity the greater harm, no matter what the outcome of the AIDS epidemic.

J.B.T.

The House by the Side Of the Road

A good many years ago I sat in the audience at a breakfast meeting and listened to a fellow physician—an anesthesiologist, as it turned out, who pioneered in obstetrical anesthesia—speak passionately of his dream of immunizing the forgotten and neglected poor of the forgotten and neglected corners of the earth in the days before such things were popular (there was as yet no "Third World"). He would do it with an invention of his that he called the "peace gun." And he did, though it is an open-ended task. It may have been even earlier, but I think perhaps the presentation I heard was made during the smallpox epidemic in Liberia in 1962, which began the crusade that led ultimately to the eradication of smallpox; in a single day a single person using the peace gun inoculated 5,000 individuals, impossible (or rather unapproachable) by any other means.

Dr. Robert Hingson was making his presentation that morning as the founder of Brother's Brother Foundation, which had taken upon itself the task of fulfilling its founder's dream. On this June 30 just past, President Reagan conferred on Dr. Hingson the 1987 International Service Award for his efforts, this just one among the many applauded in the accompanying guest editorial by one of his fellow anesthesiologists. By publishing that communication in this particular position, the *Journal* adds its own congratulations and felicitations to Dr. Hingson.

I am well aware that other significant contributions to medicine, world health, and society in general have gone unrecognized in these columns. I offer no apology, and establish no precedent. It is enough that however marvelous the achievements of others, Dr. Hingson's contribution, though surely equaled, remains in my view unsurpassed, and so the *Journal* is content to join the President of the United States in saluting Dr. Hingson as a friend to man.

J.B.T.

Guest Editorial

ROBERT A. HINGSON, M.D., Sc.D.: Former Memphis Anesthesiologist Receives Presidential Award

On June 30 President Ronald Reagan conferred upon Dr. Hingson the 1987 International Service Award for his contributions in medicine. Similar awards were given to 19 other persons for services in other areas. The ceremony took place at the White House. Dr. Hingson was honored for his contributions in four areas of endeavor: the development and use of the "peace gun" inoculator used for immunizations of more than 900 million persons around the world, development and promotion of techniques of conduction anesthesia for obstetrics in the interest of relieving pain and lessening maternal and infant mortality, alleviation of suffering among earthquake victims in Latin America in the 1970s, and distribution of corn seed to farmers in drought-stricken areas of the South. Humanitarian concerns activated through his aggressive organizational ability account for this latest of many honors bestowed upon Dr. Hingson.

Dr. Hingson was born April 13, 1913 at Anniston, Alabama, graduated in medicine from Emory University in 1938, and completed postgraduate studies in anesthesiology at the Mayo Clinic in 1941. During his early career he organized the first Department of Anesthesiology at the Jefferson Medical School in Philadelphia, and under the critical surveillance of the American Society of Obstetrics and Gynecology assisted in the development of the first Postgraduate School for Anesthesiology and Obstetrics in the world. So popular was his course in obstetrical anesthesia at Philadelphia from 1942 to 1945 that a larger institution was sought in collaboration with the Office of Surgeon-General Parran. That need led to his arrival in Memphis in 1945 where he was founder-professor of anesthesia at the University of Tennessee. There he remained until 1948.

The years at the University of Tennessee and its teaching facility at the John Gaston Hospital were bountiful, and the accomplishments in anesthesiology, especially pertaining to obstetrics, cogently relate to the Award. Here he instituted techniques of conduction anesthesia (caudal, spinal, epidural) for painless delivery. The overall objective was diminution of maternal and infant mortality, both of which were effected.

The experiences at Philadelphia and Memphis offered data for writing and publishing. The textbook, *Control of Pain in Childbirth*, by Lull and Hingson went through three editions, 1943, 1944 and 1948. While he was at Memphis the massive volume, *Pitkin's Conduction Anesthesia* (1948), of which he was co-author, was also published.

Another of the bases for consideration of his candidacy for the 1987 International Service Award was the development and use of the "peace gun." This also related to his three years in Memphis, for, as Dr. Hingson wrote about a year ago, "Significant is the fact that 700 million to date periglobally have been injected with the equipment first introduced into clinical medicine at the University of Tennessee and affiliate hospitals." This jet technique of percutaneous immunization was pioneered globally in 1962 in Liberia, then in the throes of a smallpox epidemic. Later the procedure was employed by the World Health Organization and others to help eradicate smallpox internationally. Dr. Hingson has told me that the highest rate of inoculation by one person in a single day was 5,000!

From Memphis in 1948 he went by invitation to Johns Hopkins to organize a Department of Obstetric Anesthesia. There he remained until 1951 when he became founder-professor of anesthesia at Western Reserve University in Cleveland where he remained until 1968. There were other faculty and hospital appointments in various capacities, more books, chapters of books and numerous articles pertaining to anesthesia over the years. As chairman of the Action Committee of the World Federation of Societies of Anesthesiologists and president and director of Anesthesia Educational and Relief Foundation he was extremely active in the development of three anesthesia training centers outside the United States.

Dr. Hingson's overview of human needs was not limited to those associated with anesthesia. In 1958, while at Western Reserve University, he organized and was president and director of the Brother's Brother, a non-profit foundation through which he has functioned in his delivery of welfare worldwide, using volunteer workers, including physicians. Brother's Brother is an interfaith, interracial, and interdisciplinary foundation functioning from modest surroundings in Pittsburgh at a very low overhead.

Although nominally "retired," Hingson is still active at his home in Ocilla, Georgia. He remains medical director of the Brother's Brother Foundation, in which his son Luke now functions as president. Dr. Hingson is chairman of Rotary's District 692 Polio Plus Program, which aims towards a goal of $123 million to rid the world of polio by 1995, and he is also chairman of the World Service Committee of the Ocilla Rotary Club. In his spare time he becomes involved in small tasks like planting 100,000 pine trees on his farm in southern Georgia.

Congratulations! Ad multos annos!

Charles B. Pittinger, M.D.
Professor of Anesthesiology, Emeritus
Vanderbilt University
School of Medicine—Room 3325
Nashville, TN 37232

our mail box

Liability Loss Prevention

To the Editor:

I am in the habit of reading the Loss Prevention Case of the Month in the *Journal* regularly with interest (and some trepidation) and feel that I learn from doing that.

The case report in the May issue, "Close Counts Only in Horseshoes" (*J Tenn Med Assoc* 80:296, 1987), concerning a very unusual neurological problem disturbed me greatly. First, I would like to ask Dr. Avery if a judgment was obtained in this case. He did not specifically state that in the limited space available. Secondly, if there was a judgment, I would be interested in knowing more about the basis on which the jury or court made such a decision. Dr. Avery's comments seem to imply that, even with very careful and thorough diagnostic steps, an error in diagnosis is sufficient reason for some settlement under the law.

That is different from what I have heard in the past about the law and its interpretation in Tennessee. My understanding is that the law requires "the usual standards of medical care" but does not always require a consultation in every atypical case. I do not disagree with the wisdom of obtaining consultation frequently.

Perhaps Dr. Avery could give us some more information about the outcome of this case.

James A. Burdette, M.D.
Route 6
Box 74C
Lenoir City, TN 37771

Reply

The Loss Prevention Case of the Month entitled "Close Counts Only in Horseshoes" has produced some responses that need to be addressed. In the next to the last paragraph in the Loss Prevention Notes section of the case, I indicated that if the physician had been sensitive to his patient's dissatisfaction with his management of the case, and had he called for a consultation, that litigation *could* have been avoided. As a matter of fact, what I should have said is that "litigation *might* have been avoided." Dr. Burdette is very correct in stating that the law does not require consultation in every atypical case; in fact, the law does not *require* consultation in any particular case. Dr. Burdette is again correct in stating that what the law does require is that the "usual standards of medical care are met."

The case was presented to illustrate that even in the face of an extremely careful evaluation, and a logical conclusion as to diagnosis and recommended treatment, a catastrophic outcome, such as paraplegia, can and does frequently result in loss payments to the plaintiff.

The "Cases of the Month" are taken directly from State Volunteer Mutual Insurance Company experience, though for obvious reasons, the facts of the case may be altered to obscure identification and/or to make a given loss prevention point that is believed to be pertinent to the case itself.

Very frequently the defendant physician and his insurance carrier find themselves in the position where medical reality as perceived by the defendant physician and the Claims Review Committee of SVMIC is in direct opposition to the legal reality as it is viewed by our own claims attorney at SVMIC and our doctor's defense attorney. In these situations, we very frequently find jury awards being made, and in some cases, with the consent of the insured, we find settlements to be necessary.

Some neurosurgeons have written us questioning some of the medical facts as presented in the case. Most of the questions are legitimate, and the conclusions might well have had some bearing on the case itself, but again, for obvious reasons, to protect the physician and patient confidentiality, we cannot be more specific in these case presentations.

J. Kelley Avery, M.D.
Chairman, Loss Prevention Committee
State Volunteer Mutual Insurance Co.
P.O. Box 380
Nashville, TN 37202

in memoriam

Jonathan J. Armstrong, Sr., age 93. Died June 11, 1987. Graduate of Tulane University School of Medicine. Member of Chattanooga-Hamilton County Medical Society.

James O. Hale, age 71. Died June 6, 1987. Graduate of Vanderbilt University School of Medicine. Member of Washington-Unicoi-Johnson County Medical Association.

J. Frank Manning, age 81. Died May 22, 1987. Graduate of Emory University School of Medicine. Member of Blount County Medical Society.

J. Ray Smith, age 69. Died May 22, 1987. Graduate of University of Tennessee College of Medicine. Member of Henry County Medical Society.

TMA Members Receive AMA Physician's Recognition Award

Thirty-nine TMA members qualified for the AMA Physician's Recognition Award during May 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Robert B. Adkins, M.D., Nashville
Jeanne F. Ballinger, M.D., Nashville
Jack M. Batson, M.D., Nashville
Richard T. Beeler, M.D., Nashville
Duane C. Budd, M.D., Johnson City
Thomas V. Clayton, M.D., Johnson City
Lawrence L. Cohen, M.D., Memphis
Laura L. Dunbar, M.D., Nashville
John C. Frist, Jr., M.D., Nashville
Waverly S. Green, Jr., M.D., Bristol
Radwan F. Haykal, M.D., Memphis
Laurence W. Jones, M.D., Union City
Wayne Y. Kim, M.D., Chattanooga
James C. Lett, M.D., Erin
William H. Marsh, M.D., Chattanooga
Gailen D. Marshall, M.D., Memphis
Joe M. Miller, M.D., Nashville
Hays Mitchell, M.D., Cleveland
Shamin Moinuddin, M.D., Memphis
Richard A. Orland M.D., Nashville
P. E. Orpet, Jr., M.D., Memphis
Ward C. Parsons, III, M.D., Memphis
Stewart N. Perlman, M.D., Nashville
John B. Plonk, M.D., Madison
Edward M. Reaves, M.D., Memphis
William H. Roberts, M.D., Jackson
John C. Rylands, M.D., Knoxville
Jon R. Simons. M.D., Knoxville
Homer L. Staley, M.D., Lawrenceburg
Radford C. Stewart, III, M.D., Nashville
Jack C. Stripling, M.D., Lexington
Steven M. Tate, M.D., Nashville
Steven M. Thomas, M.D., Chattanooga
John V. Webb, M.D., Maryville
Alvin J. Weber, III, M.D., Memphis
Robert B. Whittle, M.D., Knoxville
Ronald K. Wibking, M.D., Clarksville
James H. Williams, M.D., Milan
George A. Youngberg, M.D., Johnson City

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY
Howard A. Brown, M.D., Chattanooga
Gary E. Mills, M.D., Chattanooga
Jane L. Rohrer, M.D., Chattanooga

CONSOLIDATED MEDICAL ASSEMBLY OF WEST TENNESSEE
Claude S. Bridges, M.D., Trenton
David L. Garey, M.D., Jackson
Renee Harless, M.D., Jackson

MONTGOMERY COUNTY MEDICAL SOCIETY
Edwin C. Glassell, M.D., Clarksville

NASHVILLE ACADEMY OF MEDICINE
Paul B. Batalden, M.D., Nashville
Ernest Andrew Bell, Jr., M.D., Madison
Edward Neal Brin, M.D., Nashville
Michael Donahue Decker, M.D., Nashville
Lewis B. Lefkowitz, M.D., Nashville
Paula S. Nunn, M.D., Nashville
Kenneth Lewis Sears, M.D., Nashville
William Alfred Shell, Jr., M.D., Nashville
Richard S. Stein, M.D., Nashville
Thomas Philip Toomey, M.D., Nashville

(Students)

Mark Allen Dosmann, Nashville
Alex H. Kim, Nashville

NORTHWEST TENNESSEE ACADEMY OF MEDICINE
Stevens D. Melton, M.D., Dyersburg

WASHINGTON-UNICOI-JOHNSON COUNTY MEDICAL ASSOCIATION
Terry C. Borel, M.D., Johnson City

WILLIAMSON COUNTY MEDICAL SOCIETY
Kenneth Brenton Dodge, M.D., Franklin
Michael D. Langford, M.D., Franklin

announcements

CALENDAR OF MEETINGS

NATIONAL

Sept. 9-12	American Society for Surgery of the Hand—Convention Center, San Antonio, Tex.
Sept. 12	American Rhinologic Society—Las Vegas
Sept. 14-17	American Academy of Family Physicians—Fairmont, San Francisco
Sept. 14-19	American Electroencephalographic Society—Adam's Mark Hotel, St. Louis
Sept. 16-19	American Thyroid Association—J. W. Marriott, Washington, D.C.
Sept. 18-19	American College of Nuclear Medicine—Shoreham Hotel, Washington, D.C.
Sept. 19-23	American Academy of Otolaryngology-Head and Neck Surgery—Chicago
Sept. 22-24	American Paraplegia Society—Riviera Hotel, Las Vegas
Oct. 6-9	American Institute of Ultrasound in Medicine—Sheraton, New Orleans
Oct. 7-10	American School Health Association—Hyatt Regency, Indianapolis
Oct. 8-9	Chicago Lung Conference on Asthma (sponsored by Chicago Lung Association and Chicago Thoracic Society)—Chicago
Oct. 8-10	International Conference on Drug Research and Immunologic and Infectious Disease, Antifungal Drugs: Synthesis, Preclinical and Clinical Evaluation (sponsored by NY Acad Sci)—Garden City Hotel, New York
Oct. 8-11	Central Neuropsychiatric Association—Holiday Inn at Union Station, Indianapolis
Oct. 9-12	Scientific Conference of the International Federation of Psychoanalytic Societies—New York
Oct. 10-13	International Symposium on Calcium Antagonists: Pharmacology and Clinical Research (sponsored by NY Acad Sci)—New York
Oct. 10-14	American Society of Anesthesiologists—Atlanta
Oct. 11-16	American College of Surgeons—San Francisco Hilton
Oct. 12-14	Society for Medical Decision Making—Sheraton Society Hill Hotel, Philadelphia
Oct. 15-18	American Association of Electromyography and Electrodiagnosis—Hyatt Regency, San Antonio, Tex.
Oct. 15-18	American Society of Internal Medicine—J. W. Marriott, Washington, D.C.
Oct. 18-21	American Neurological Association—Hilton & Towers, San Francisco
Oct. 18-22	American Public Health Association—New Orleans
Oct. 18-22	International Health Society—New Orleans
Oct. 18-23	American Academy of Physical Medicine and Rehabilitation—Marriott World Center, Orlando, Fla.
Oct. 19-23	American Academy of Occupational Medicine—Hyatt Regency, San Francisco
Oct. 20-25	American Academy of Neurological and Orthopaedic Surgeons—Alexis Park Resort Hotel, Las Vegas
Oct. 21-25	American Academy of Child Psychiatry—Sheraton, Washington, D.C.
Oct. 22-24	Child Neurology Society—Vacation Village, San Diego
Oct. 23-29	American Society for Therapeutic Radiology and Oncology—Sheraton, Boston
Oct. 25-28	American Association for the Study of Liver Disease—Marriott, Chicago
Oct. 25-28	Medical Group Management Association—Sheraton, Washington, D.C.
Oct. 25-30	Congress on Neurological Surgeons—Convention Center, Baltimore
Oct. 26-28	American College of Gastroenterology—Fairmont Hotel, Dallas
Oct. 26-30	American College of Chest Physicians—Marriott, Atlanta
Oct. 27-Nov. 1	American Medical Women's Association—Buena Vista Palace, Orlando, Fla.

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Knowledge and Attitudes of Memphis Parochial School Adolescents About the Acquired Immune Deficiency Syndrome

TANJA K. KONETZNY; ANNA H. KONETZNY; and LINDA L. PIFER, Ph.D.

Introduction

In the absence of a vaccine against human immunodeficiency virus (HIV), the etiologic agent of the acquired immune deficiency syndrome (AIDS), or even the immediate likelihood of developing such a vaccine, education may prove to be the first and most important line of defense against spread of the disease in persons of all ages. The recommendation of Surgeon General Koop urging immediate AIDS education of youth has been widely publicized.[1]

With the exception of those with concerns that such educational efforts will encourage sexual experimentation and/or promiscuity, reaction to the educational approach has been positive. In view of the morbidity and absolute mortality of AIDS, it is critical that all means be employed to protect youth against this modern plague. At present, education appears to offer the best solution.

Without some overall concept of the level of accurate knowledge possessed by adolescents concerning AIDS, however, it is impossible to plan appropriate educational programs. The extent of naivety and erroneous information about AIDS among adolescents must first be assessed to ensure the effectiveness of any proposed educational efforts. It is for that purpose that we undertook the present study.

Methods

One hundred seventh and eighth grade students of both sexes (median age, 13.5 years) were anonymously interviewed by means of printed, self-administered questionnaires concerning their factual knowledge, attitudes, and beliefs about AIDS. The questionnaire was essentially the same as one recently employed in a study of adolescents in the high AIDS-risk Bay Area of San Francisco.[2]

The survey was conducted during a one-week period in urban Memphis in January 1987 among students enrolled at St. Louis Catholic School, a private parochial school in Memphis. Aside from a high proportion of students belonging to the

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Roman Catholic faith, the students completing the questionnaire represented an ethnic and economic cross-section of the community.

Results

The responses made to each statement on the questionnaire designed to test the students' knowledge about AIDS are given in Table 1.

Twenty percent of the students interviewed were not aware that AIDS is "a medical condition in which the body cannot fight off diseases," and 38% were unaware that AIDS is caused by a virus. Seventeen percent believed that AIDS is transmissible by kissing, and 7% to 10% believed that "just being around" someone with AIDS was sufficient to transmit the disease. Fourteen percent did not believe or did not know that "anybody can get AIDS." Twenty-one percent were misinformed, or at best were uncertain, about the facts concerning food as a potential source of AIDS.

Concerning the possible relationship between venereal diseases and AIDS, 68% either believed that AIDS and sexually transmitted diseases were "caused by the same virus" or were uncertain as to the correct response. Sixty-three percent were wrong or unclear about knowledge concerning the cause of AIDS.

With regard to the mortality of AIDS, 91% to 95% correctly perceived AIDS as a deadly disease, but 35% did not realize that "there is no cure for AIDS" and that to date, "AIDS (cannot) be cured if treated early." Regarding prevention, only 20% knew that there is at present no vaccine for AIDS, and 10% thought that "exercising regularly" would be beneficial in avoiding AIDS.

The highest percentage of accurate scores was achieved, in descending order of accuracy, on questions 22, 21, 23, 17, 19, and 18, all of which

TABLE 1

SUBJECT RESPONSES FOR KNOWLEDGE STATEMENTS

Statement	Percent		
	True	False	Don't Know
1. AIDS is a medical condition in which your body cannot fight off diseases.	80	2	18
2. AIDS is caused by a virus.	62	23	15
3. AIDS is a condition you are born with.	9	79	12
4. Stress causes AIDS.	1	85	14
5. If you kiss someone with AIDS, you will get the disease.	17	55	28
6. If you touch someone with AIDS, you can get AIDS.	10	81	9
7. All gay men have AIDS.	4	89	7
8. What you eat can give you AIDS.	7	79	14
9. Anybody can get AIDS.	86	5	9
10. AIDS can be cured.	9	42	49
11. Women are more likely to get AIDS during their period.	4	7	89
12. AIDS can be spread by using someone's personal belongings like a comb or hairbrush.	12	74	14
13. AIDS is not at all serious; it is like having a cold.	3	94	3
14. AIDS is caused by the same virus that causes VD.	10	32	58
15. The cause of AIDS is unknown.	42	37	21
16. Just being around someone with AIDS can give you the disease.	7	82	11
17. Having sex with someone who has AIDS is one way of getting it.	94	0	6
18. If a pregnant woman has AIDS, there is a chance it may harm her unborn baby.	87	1	12
19. Most people who get AIDS usually die from the disease.	91	3	6
20. You can get AIDS by shaking hands with someone who has it.	2	88	10
21. Receiving a blood transfusion with infected blood can give a person AIDS.	97	0	3
22. You can get AIDS by sharing a needle with a drug user who has the disease.	98	0	2
23. AIDS is a life-threatening disease.	95	0	5
24. People with AIDS usually have lots of other diseases as a result of AIDS.	56	15	29
25. All gay women have AIDS.	4	81	15
26. There is no cure for AIDS.	65	12	23
27. I can avoid getting AIDS by exercising regularly.	10	60	30
28. AIDS can be cured if treated early.	8	63	29
29. A new vaccine has recently been developed for the treatment of AIDS.	26	20	54

relate to modes of HIV transmission or to the seriousness of AIDS.

Ninety-eight percent were aware of the AIDS-intravenous drug connection, and 97% knew that AIDS can be contracted via blood transfusion. Ninety-five percent knew that AIDS is life-threatening, and 91% believe that most people with AIDS die of the disease. Ninety-four percent realized that having sex with a person with AIDS can transmit the disease, and 87% knew that if a pregnant woman has AIDS, "there is a chance that it may harm her unborn baby."

The students' responses relative to their attitudes and beliefs about AIDS are recorded in Table 2. Seventeen percent doubted that AIDS is "as big a problem as the media suggest." Twenty-six percent were "not worried" about getting AIDS, but only 65% indicated that they were "not the kind of person who is likely to get AIDS." Twenty-one percent believed that they had "heard enough about AIDS" and did not "want to hear any more about it," but 86% thought it important that students learn about AIDS in Family Life Education classes. Twenty-two percent stated that they had received instruction about AIDS as part of their school curriculum.

Memphis adolescents' attitudes and beliefs about AIDS were remarkably similar to those of Bay Area teenagers on Questions 1, 3, 4, 6, and 9. The greatest difference in response was noted in Question 7, which asked whether or not students would take "a free blood test to see if you have the AIDS virus." Only 10% of Memphis students, as opposed to 51.5% of San Francisco adolescents, would take such a test.

One other noteworthy difference was seen in Question 10 of Table 2, which inquired whether or not the student interviewees had received any formal instruction about AIDS in their school curricula. Only 35.3% of San Francisco and 22% of Memphis adolescents had.

Discussion

With regard to overall knowledge about AIDS, the Memphis parochial school adolescents outperformed the older adolescents from the high AIDS-incidence Bay Area of San Francisco, who were the subjects of a similar recent report.[2] On questions based on factual information about AIDS (Table 1), Memphis adolescents (median age, 13.5 years) scored significantly higher than the older California teenagers on 18 of 29 (62%) questions.

Factors that may have influenced the results include (1) the time lag between the two surveys (20 months), (2) difference in age (the California students were, on an average, three years older than their Memphis counterparts), (3) differences in socioeconomic background, and (4) that the Memphis adolescents attended a Roman Catholic parochial school, while those in San Francisco attended ten public high schools.

Although students from both cities had good concepts as to how AIDS is actually transmitted, fundamental knowledge concerning the viral etiology of AIDS, the prognosis of the AIDS patient, and the availability of treatment and a vac-

TABLE 2

SUBJECT RESPONSES FOR ATTITUDES AND BELIEFS ABOUT AIDS

Statement	Percent		
	True	False	Don't Know
1. AIDS is not as big a problem as the media suggest.	7	83	10
2. I am afraid of getting AIDS.	73	24	3
3. I am not worried about getting AIDS.	26	68	6
4. I am not the kind of person likely to get AIDS.	65	16	19
5. I am less likely than most people to get AIDS.	44	28	28
6. I'd rather get any other disease than AIDS.	55	17	28
7. If a free blood test were available to see if you have the AIDS virus, would you take it?	10	67	23
8. I've heard enough about AIDS and I don't want to hear any more about it.	21	70	9
9. It it important that students learn about AIDS in Family Life Education classes.	86	3	11
10. Have you had any instruction about AIDS in your school curriculum?	22	62	16

cine were decidedly lacking. Another knowledge deficit was underscored by the observation that a substantial number of students in both groups were concerned that AIDS may be acquired by casual contact, regardless of the lack of data to support such a belief.

Although AIDS has received unprecedented attention via the lay press, television, and radio, it is clear from the results of our study and the one from San Francisco conducted nearly two years ago that adolescents are not receiving adequate correct information about AIDS. Cursory examination of the data suggests that these adolescents possess good general knowledge about AIDS, but a closer inspection reveals that there are dangerous information gaps. Though the news media have done a fairly good job of reporting the emerging AIDS crisis, the information has often been sensational, superficial, and occasionally misleading.

Estimates by the Centers for Disease Control and the National Institutes of Health, based upon the actual number of HIV antibody-positive individuals at the present time, suggest that by 1991, 270,000 clinical cases of AIDS will have been reported. CDC-NIH estimates also suggest that 1 million to 1.5 million people are now probably infected with the AIDS virus. In view of these grim projections, it is unconscionable that only 22% to 35% of our adolescents have ever received formal instruction about AIDS.

Since 92% to 96% of AIDS cases reported to date have been linked to specific behavioral features, i.e., male homosexual activity (75% to 79%) and intravenous drug abuse (17% to 19%), it is clear that specific education might logically be expected to contribute substantially to halting the spread of AIDS among both adolescents and adults. In order that our children may be protected from the spread of AIDS, education concerning abstinence and safer sexual practices must be supported by the medical/scientific community. Sensitivity to the moral values of children and their parents is essential in order that traditional objections to value-free "sex education" be satisfactorily resolved. At present, it would seem that education is the best vaccine. We have a professional and moral obligation to dispense it liberally, with extreme accuracy, and with sensitivity to the moral, social, and ethical issues involved.

Acknowledgments

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Retroperitoneal Cyst in a 4-Year-Old Child

A Case Report

GERALD R. JERKINS, M.D. and JEFF D. WHITFIELD, M.D.

Introduction

Retroperitoneal cysts are rare and may present themselves in a variety of ways, including symptoms related to size and compression of adjacent structures. We report herein the case of a 4-year-old child with abdominal pain who was found to have a large retroperitoneal cyst lined with gastric mucosa in the upper pole of the right kidney.

Case Report

A 4-year-old white boy was seen by his local physician for abdominal pain that had been present for several weeks and had become more intense and localized to the right upper quadrant. Physical examination was negative and urinalysis and serum electrolytes were normal. An intravenous urogram revealed a large mass in the upper pole of the right kidney, and computed tomography (CT) demonstrated it to be a fluid-filled cyst (Fig. 1). On referral of the patient for further evaluation, a renal scan demonstrated virtually no blood flow to the mass and no evidence of functioning renal tissue about it (Fig. 2).

At cystoscopy there was no evidence of a duplicated ureter on either side, and a retrograde ureterogram on the right revealed a single ureter with a downward displacement of the collecting system (Fig. 3). Exploration through a right anterior subcostal incision revealed a fluid-filled cyst adherent to the liver, diaphragm, and upper pole of the right kidney. There was no evidence of renal duplication. In its greatest dimension the cyst was larger than 10 cm and was filled with cloudy, brown fluid. Part of the cyst lining was irregular and firm, and a frozen section revealed gastric mucosa with ulceration. Intraperitoneal exploration showed no connection between the mass and any intraperitoneal structure, including the stomach and intestine.

The patient had an uneventful postoperative course. Two months following surgery he was reevaluated for persistent abdominal pain. A repeat CT scan demonstrated no renal obstruction or recurrence of the cyst, though small cysts were noted within both kidneys and the head of the pancreas. The patient is presently stable, without evidence of enlargement of any of the cysts.

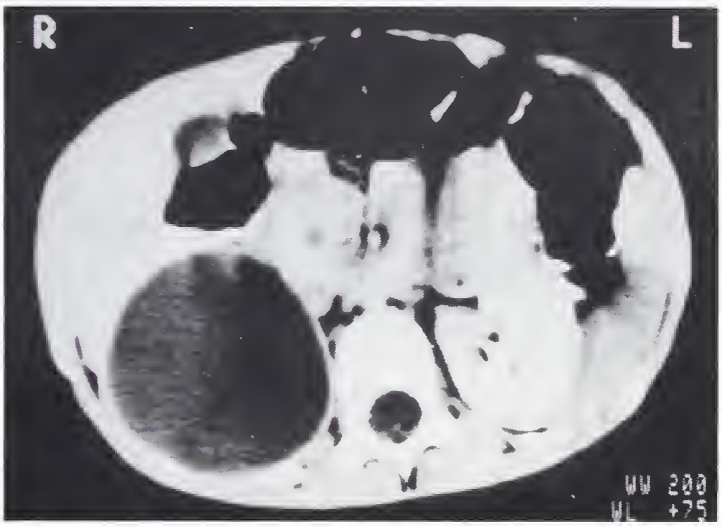

Figure 1. CT scan demonstrating a large right retroperitoneal cyst.

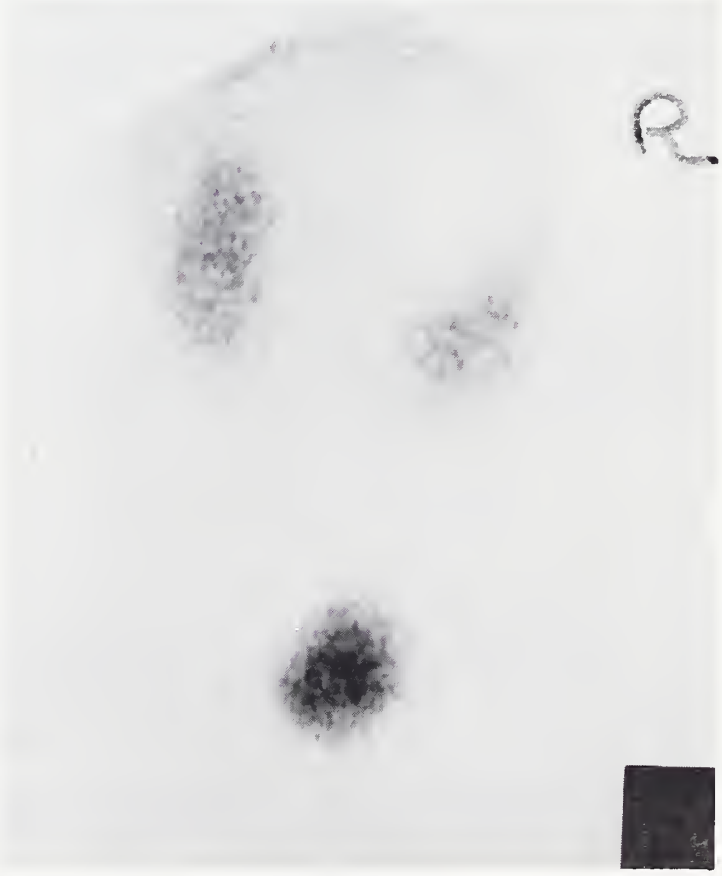

Figure 2. Radionucleotide renal scan indicating no significant blood supply or functioning renal tissue related to the mass.

From the Department of Pediatric Urology, University of Tennessee, Memphis.

Reprint requests to Department of Pediatric Urology, University of Tennessee College of Medicine, 848 Adams Ave., Suite 403, Memphis, TN 38103 (Dr. Jerkins).

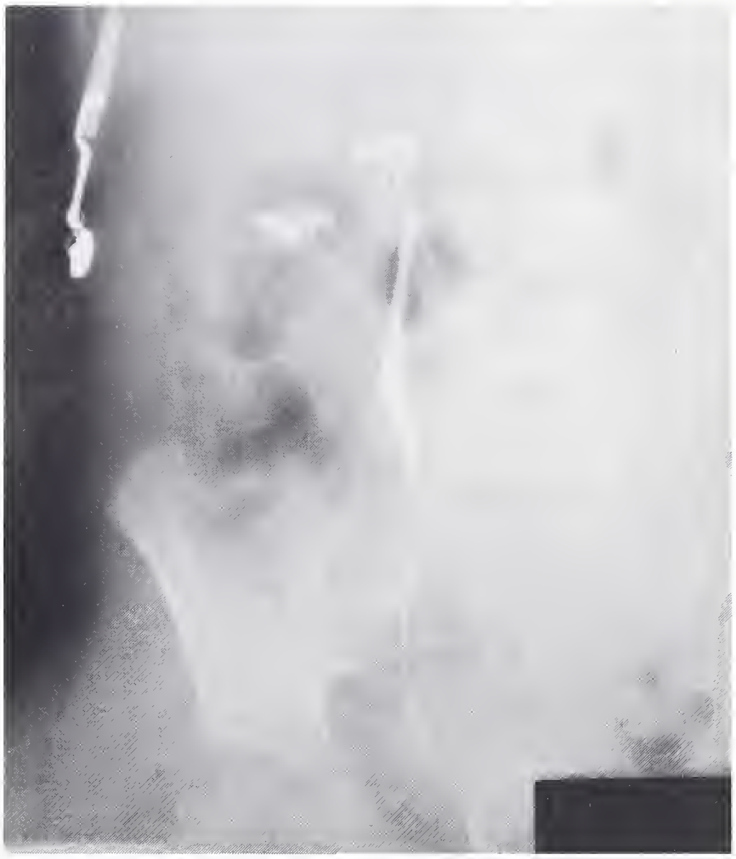

Figure 3. Retrograde pyelogram demonstrating displacement of right renal collecting system.

Discussion

Primary tumors of the retroperitoneum are rare, and interest the urologist because of their potential for compression or compromise of the urinary tract. Tumors can arise from mesodermal, neural, urogenital, and other embryonic remnants. Though the majority of such retroperitoneal tumors are malignant, benign masses or cysts may arise from the same tissue, though less frequently.[1]

Cysts compose only a small proportion of retroperitoneal masses. They are commonly benign and slow growing, and become symptomatic as a result of size and pressure on adjacent organs.[2] This patient apparently became symptomatic because of bleeding into and expansion of the cyst.

Gastric duplications are rare, with fewer than 90 cases reported in the literature. The majority were diagnosed before 1 year of age, with only 14% noted between ages 1 and 12 years. Such duplications can manifest themselves in a variety of ways, including symptoms related to the gastrointestinal tract or compression of other retroperitoneal structures. As in this case, communication between the cyst and the remaining gastrointestinal tract is usually not seen.[3]

The follow-up studies in this child suggest a remaining rest of similar tissue in the kidney and pancreatic area. Long-term follow-up will be necessary for any progressive enlargement of the remaining cyst.

Though as with any intra-abdominal mass, retroperitoneal masses should be thoroughly evaluated prior to surgical exploration, most of these lesions will not be accurately identified without surgical exploration and excision. Preoperative evaluation with ultrasonography and a CT scan are extremely useful for defining the mass and its relationship with surrounding structures. The value of the thorough preoperative evaluation is in determining the best surgical approach for removal of the mass.

This case represents an unusual presentation of a rare type of retroperitoneal cyst, and underscores the need to consider retroperitoneal cysts in the differential diagnosis of any large intra-abdominal mass displacing the kidney and ureters.

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Pruritus Due to Infected Pancreatic Duct Catheter

MELVIN L. ELSON, M.D. and ANTONIO M. GRANDA, M.D.

Introduction

Itching is one of the most common presenting symptoms in the dermatologist's and internist's office. The causes are myriad[1] and though most of the time the etiology is easily determined and easily corrected, some of the time there is a serious underlying cause that must be determined before the patient can obtain relief. This is the case report of such a patient.

Case Report

A 71-year-old white woman was seen on April 29, 1986 with a history of itching for six months. She had seen three physicians, who treated her with topical and systemic steroids, and antihistamines, all without relief. Routine laboratory studies, including complete blood cell count, differential, and routine chemistries, were within normal limits. The patient was a lover of and frequently cared for neighborhood dogs and had them in her house.

Although mites could not be demonstrated by potassium hydroxide, with this history and the fact that the itching was significantly more severe at night, the patient was treated with a course of crotamiton (Eurax) lotion, which initially seemed to help. Within a week, however, her itching returned and she was most uncomfortable. She was admitted on May 13, 1986 to St. Thomas Medical Center for evaluation of intractable itching.

Past history was significant only for a number of surgical procedures: A cholecystectomy in the 1940s, T&A in 1950, hemorrhoidectomy in 1981, a common duct stone in 1981 that required surgery, and a stone in the pancreatic duct in 1985 for which she again had surgery.

Physical examination revealed an elderly obese white woman who was constantly scratching; her blood pressure was 150/82 mm Hg, pulse 86/min, respirations 18/min, and temperature 98.6°F orally. Skin examination revealed erythema with multiple excoriations over the skin surface and purpura over the extensor aspects of the arms. The rest of the physical examination was within normal limits except for obesity and a number of surgical scars of the abdomen.

Laboratory work revealed negative stool guaiac, normal electrolytes, glucose 104 mg/dl, SGOT 12 U/L, alkaline phosphatase 119 U/L, bilirubin 0.3 mg/dl, protein 5.5 gm/dl, albumin 3.6 gm/dl, amylase 70 U/dl, lipase 7 U/L, uric acid 4.9 mg/dl, cholesterol 213 mg/dl, BUN 19 mg/dl, creatinine 1.2 mg/dl, WBC count 8,100/cu mm, with normal differential, hematocrit 28.5%, and iron 28 μg/dl with a total iron binding capacity of 396 μg/dl.

Because of the number of gastrointestinal procedures performed in the past, a persistent diarrhea that developed while the patient was hospitalized, and the anemia, we decided to look more closely at the GI tract, despite negative stool guaiacs on a number of occasions. Ultrasonography of the abdomen revealed status postcholecystectomy, and CT revealed postoperative changes including biliary tract air and a pancreatic duct catheter.

On consultation with the surgeons who performed the patient's surgery the previous year it was decided that since the catheter should have dropped into the bowel and been expelled, but had not, that it should be removed, and on May 19, 1986 it was removed endoscopically. Pathologic examination revealed "masses of filamentous rods and cocci adherent to and obstructing the lumen of the catheter," with bleeding from the bowel around the catheter.

Following this procedure, the patient was discharged and placed on chlorpromazine (Thorazine) 50 mg each night at bedtime, perphenazine and amitriptyline (Triavil 2-10) three times a day for itching, topical steroids, ranitidine (Zantac) 150 mg two times a day, a ferrous sulfate (Feosol) spansule daily, and prednisone 20 mg every morning.

Over the next two months her hematocrit rose to 36.5%, her itching ceased, and it was possible to taper her off all medication. By the end of the year, the patient's skin was completely healed and she had no problem with itching. She was last seen in March 1987, still asymptomatic.

Discussion

Here we have an elderly woman who confronted us with a very common problem that all practicing physicians see every day, i.e., itching. Most of the time, the etiology is easily determined and the patient does rather well with topical emollients and antihistamines or similar medication, but in this instance the etiology was not readily determined, and it was necessary to be detectives to determine the etiology. Many things have been related in the past to itching, especially in the elderly,[2] and it would serve us well to keep in mind that chronic infections should be sought in patients who do not readily respond to conservative therapy, and that sometimes these infections can be quite difficult to find.

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Special Item

Inaugural Address of the AMA President

WILLIAM S. HOTCHKISS, M.D.
President—American Medical Association

I come before you tonight with feelings of pride, humility, enthusiasm, resolve, and deep respect. I respect the many outstanding physicians who have led the American Medical Association before me and each of you for your selfless commitment in working on behalf of the health of the American people. But most of all, I respect the institution of medicine, and what it stands for—health, dignity, service, and excellence.

For more than 100 years, the AMA has dedicated itself to preserving those fundamental ideals of American medicine, and I'm proud to be a part of that tradition. I like to remind people that the AMA is a caring organization of service—service to the American people.

As physicians, we are not here to do battle with the government for our self-interests; we are not here to become leaders of corporate America; we are certainly not here to amass wealth. We are here for one reason, and that is to take care of sick people; that is what our patients want and deserve.

They are concerned about costs, especially hospital costs, but quality is the *sine qua non.* There is no question that medical care in the United States is the best in the world today—better than at any time in history. It is also worth noting that this was not always the case.

In the decades around the turn of the century, European medicine was generally considered to be the best. It was customary at that time for American doctors, after they had learned as much as they could here, to spend a year or two in Europe. When they returned, they were recognized as the best physicians in our country. Isn't it amazing that thousands of doctors from all over the world now come to America to learn our techniques and methods of treatment? Most of them have been so impressed that they have, to the extent allowed by law, chosen to remain in this country and practice here for the rest of their lives.

This wonderful, productive, inventive, free-enterprise system of medical care is a heritage from our professional forefathers, and it is the obligation of our generation to protect it and further it to the best of our ability. That takes the honesty to correct the system when things cease to function.

That's why I'm so deeply concerned about the Medicare program. Certainly from the standpoint of making care available to many elderly patients, who might otherwise not have sought and received it, the program must be judged an unqualified success, but there are major problems ahead. As currently structured, it is estimated that Part A of the trust fund will be bankrupt by the year 2002. By the year 2017 it will be in debt $1 trillion. Finally, we should all note that by the year 2010, U.S. entitlement programs will consume two-thirds of the federal budget.

This grave scenario doesn't have to happen. We have a commitment to our elderly and to our health care system. One year ago, we publicly demonstrated that commitment by proposing massive restructuring of the Medicare system which would convert it to a sound actuarial basis. As patient advocates we will continue to lobby for adequate funding for this vital program.

What can we do as leaders and as practicing physicians to protect and further our free-enterprise system of medical care, which has served

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Presented as the Inaugural Address at the Annual Meeting of the American Medical Association, Chicago, June 24, 1987.

the public so well? Above all other things, we must be patient advocates in every sense of the word, and that means offering only the highest quality of medical care to each individual. In hospitals, we must insist that clinical decisions ultimately fall to physicians. In HMOs and other new entities that fall beneath the rubric of corporate medicine, physicians must refuse to walk a fine line between the bottom line and patient care. We must refuse to buckle to shameful government pressures to deliver less than thorough care, simply so that Uncle Sam can balance the budget on the backs of the elderly or the poor.

Our high standards of care must be protected through vigorous peer review programs. When we show a steadfast intolerance for mediocrity or worse among our peers, we are being patient advocates. I am proud of the AMA's role in promoting peer review programs that work. We were instrumental in supporting new antitrust provisions that make it possible to engage in peer review, without living in fear of legal retribution. And we were victorious in convincing the government that current PRO procedures denied physicians due process of the law. The entire process has now been amended, as you know, and while it isn't perfect, the result is a system that will work for patients and their doctors.

Patient advocacy means putting forth intensive efforts to eradicate threats to public health—and the AMA has a long and active history in this mission. Each of us has a tremendous capacity to inform and to educate, and our profession calls on us to be selfless and generous in that role and with our time. But today there is one especially compelling reason why each of us must give of ourselves completely to others; that reason is AIDS.

If you have not been touched by the grim spectre of AIDS through the death of a friend, a neighbor, a relative, or a business associate, you very likely will—and soon. The U.S. Public Health Service estimates that approximately 1.5 million U.S. citizens are infected with the AIDS virus at this time, and that by 1991 we will see 270,000 cases of AIDS in our country. Since the disease carries a 100% mortality, the death toll by that time will approximate the total loss of military personnel suffered by the United States in World War II.

I'm proud of the action taken by our House of Delegates yesterday, after thoughtful consideration, on the Board Report on AIDS. I certainly recognize that our action is only a first step in making recommendations for Americans—but it is an important step. We must use every opportunity that presents itself to educate and inform the public and the profession and to encourage scientific research with adequate government funding to control this epidemic.

We must think of our children, and it isn't just because of AIDS. Last year the AMA published a White Paper on Adolescent Health. The dark statistics contained in that report cut deeply, and devastate all who read them, and well they should. They describe a life of hell for hundreds of thousands of adolescents. They report the dismal story of 5,000 teenagers who were despairing so greatly that they took their own lives last year.

Think of the 900,000 teenagers who roam the streets as working prostitutes. Think of the 1 million school-aged girls who will become pregnant this year, and think of their lives, their hopes, and dreams. Think of the welfare of the children of those children.

Tonight I am calling on all physicians to use more of their hearts in patient care. Advocacy means treating all patients with the courtesy and respect that they deserve. This message is impossible to convey to them when we ask patients to wait weeks for an appointment, or hours in the reception area, especially if we spend only a few minutes in the consultation and examining room with them.

We must be honest enough to face the unfortunate fact that a few physicians are not fit to practice medicine and must be culled from the system. The AMA is actively engaged in this effort. Our physician masterfile prevents a doctor from being licensed in one state after having a license revoked in another. But certainly, the overwhelming majority of physicians embrace excellence and I think that's because our profession has traditionally attracted great people.

Being a physician is just as thrilling today as it was 30 years ago. It is the most rewarding of all professions. Yes, there are different methods of delivery of care and there are more regulations. But there is always the opportunity to serve our patients. And medicine—which we study all of our lives—is and always will be fascinating. We must encourage good students to enter the medical field and this lifelong profession of learning. Young physicians can anticipate an exciting career. With increasing and improving technology, their opportunity to render service and return sick patients to good health will far surpass anything our generations have enjoyed.

As a physician, I'm proud of the AMA's accomplishments; I'm proud of our "can do" attitude. In the 140-year history of the AMA, we've cleaned up the water supplies, we've facilitated a national system of local and state health departments, and we've been staunch advocates of seatbelt legislation and helmet laws. Today, we're engaged in a war against AIDS and another battle against tobacco-related diseases.

Truly, the AMA is an organization on the move. It's active, decisive, and tough, and it speaks for all physicians. The AMA believes, as I do, that medicine is a helping profession. Our mission is one of patient advocacy, and one of upholding a standard of excellence that has come to be the envy of the rest of the world.

I call on each of you tonight to encourage our fellow physicians to renew their commitment to American medicine as a helping profession—a caring profession. That is our strength, and that is the salvation of high quality care for the people of our great country.

As individual physicians, our strength is limited. To illustrate the importance of working together, I close with the first verse of Kipling's "Law of the Jungle."

> Now this is the Law of the Jungle—As old and as true as the sky: and the wolf that shall keep it may prosper, but the wolf that shall break it must die. As the creeper that girdles the tree-trunk, the law runneth forward and back . . . for the strength of the pack is the wolf, and the strength of the wolf is the pack.

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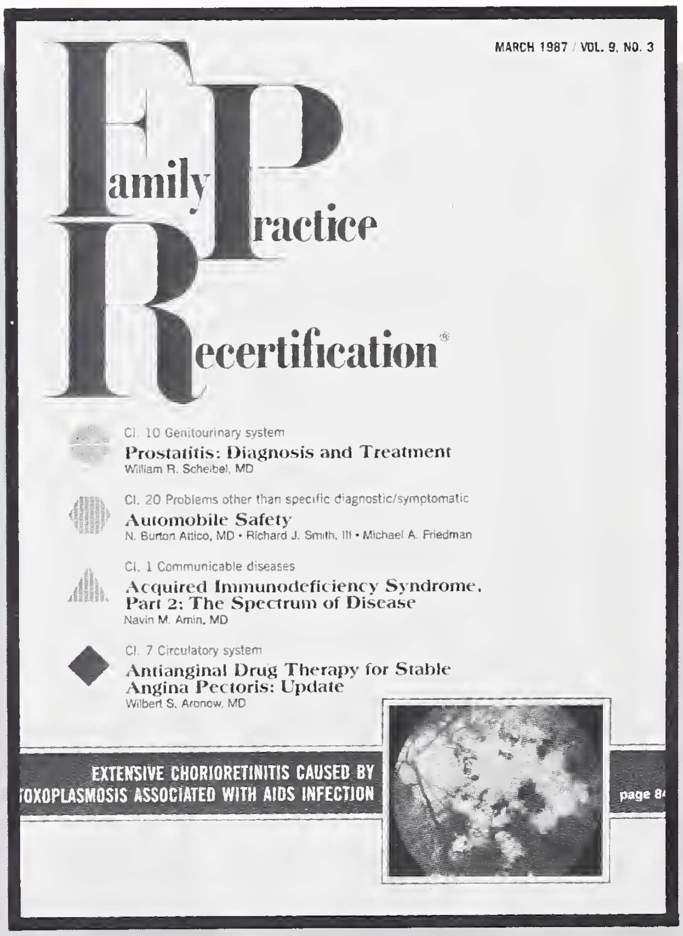

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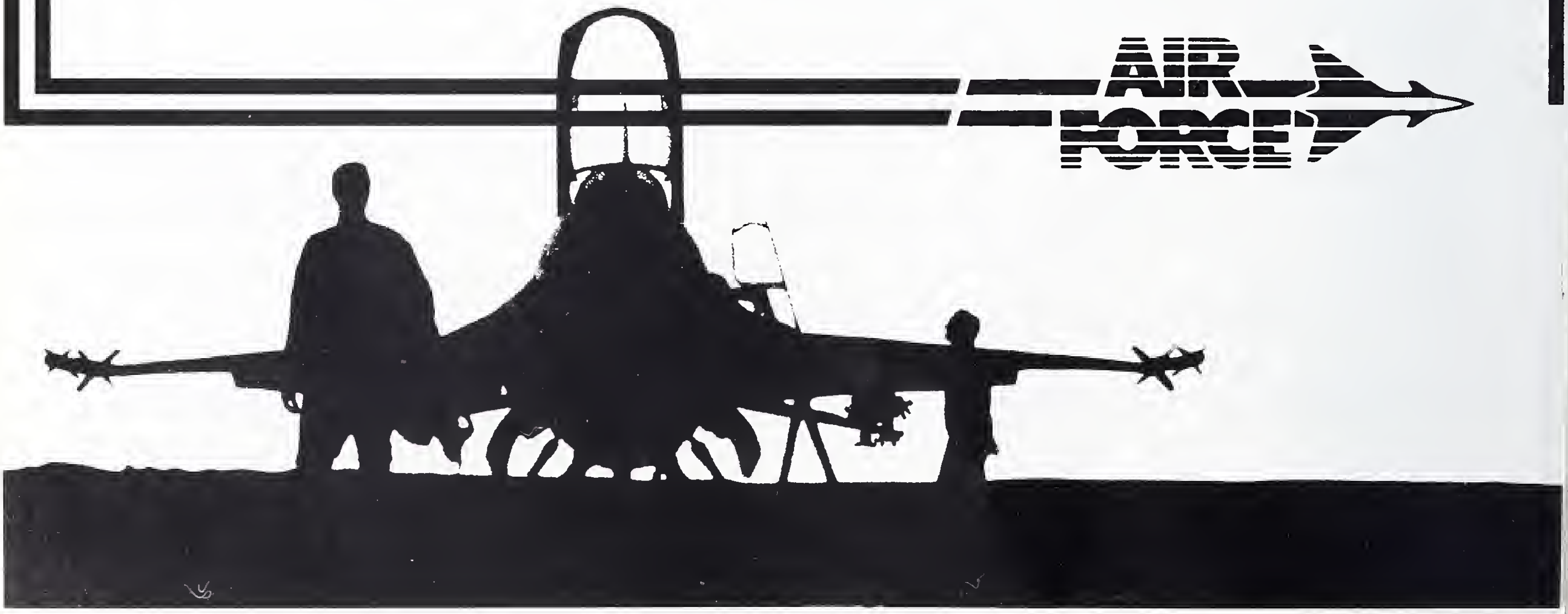

Prevention and Control of AIDS

An Interim Report

Introduction

Responding sensitively, intelligently, and effectively to the growing AIDS crisis is one of the crucial public health problems facing the nation. Prevention and control of the disease must be an essential part of that response because there is, at present, no known cure for AIDS patients.

Recommendations in this report have as their foundation an overriding concern for a judicious balance between the well-being of HIV-positive patients and the protection of the public health. These recommendations are based upon the best information and data available at present. The AMA will continuously monitor and analyze developments in AIDS and update AMA policy and recommendations as dictated by advances in knowledge.

Education continues to be the major weapon against spread of HIV infection. Physicians should assume the leadership role in educating themselves, their patients and the public. Individuals in society also must assume responsiblity for being well informed and for actions that affect their own health and the health of others. In developing this report, the Board emphasizes the need for concerted and cooperative efforts by all members of society in the fight against AIDS. The recommendations outlined below are designed to help in successfully confronting this challenge to society's well-being.

Background

The Current Climate

It is estimated that 5 million to 10 million people are infected with HIV virus worldwide. AIDS has been reported in more than 100 countries. In the United States HIV-infected individuals may number 1.5 million, approximately 35,000 of whom have been reported to suffer from AIDS and more than 20,000 of whom are dead.

The U.S. Public Health Service has projected that by 1991 there may be 323,000 reported patients with AIDS and as many as 200,000 of them may be dead by that time. In addition, conversion rates of seropositive people to AIDS status now appear to be higher than early preliminary estimates. Originally under 20% were thought to convert. It now appears that, without treatment advances, a much higher percentage will develop the disease.

Seventeen percent of the AIDS cases have been intravenous drug abusers, 66% have been homosexual/bisexual men, 8% have been homosexual male IV drug users; female, heterosexual male, and pediatric victims infected by the transfusion of blood or blood products, sexual contact, or prenatally in the case of infants, account for the bulk of the balance.

Polls indicate that AIDS has become the highest priority health concern of the American public, ahead of heart disease and cancer. It has already caused changes in a variety of public attitudes. Sexual abstinence, monogamous relationships, and the use of condoms are being widely promoted in the media by public officials and many private organizations. IV drug abusers are being counseled to use clean needles and to avoid sharing needles. Education on the sexual transmission of the AIDS virus is being extended to school children. The nation is more sensitive to the rights of those afflicted with the disease to be free from discrimination, regardless of the manner by which they became infected.

Historical Control Measures for Infectious Diseases

A primary mode of transmission of AIDS is through sexual contact, and the control efforts for sexually transmitted diseases (STD) that have been instituted in the past are sources of analogies for prevention and control of AIDS. National programs to control STDs were established during the beginning of World War I. For the following 50 years the focus was almost exclusively on the control of syphilis and its complications. During World War II rapid treatment centers for syphilis and gonorrhea were established. Public health officials instituted limited contact-tracing, had the authority to close sex bars and clubs, to order tests for prostitutes, and, most importantly, had effective therapy to offer. Widespread availability of penicillin led to the dissolution of the rapid treatment centers and of the clinical specialty, syphilology. Every state in the Union at one time required all persons seeking marriage licenses to be tested for syphilis. During the 1950s and 1960s federal assistance programs continued to support contact-tracing, serological screening, and patient education.

In the late 1960s public health officials were concerned about the rapidly escalating cases of gonorrhea, and projects were instituted to increase case-finding and contact-tracing. In 1972 financial assistance for STD

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control by the federal government was dramatically increased and by 1982 gonorrhea accounted for nearly three-fourths of the federal STD dollar. During the 1970s gonorrhea control efforts evolved through overlapping phases that included objectives to lower disease incidence and the occurrence of drug-resistant bacteria, focused screening on high-risk patients, intensified follow-up of treatment failures, and used patient counseling as a means of increasing compliance with therapy and improving contact-tracing. The latter was deemed especially important since the large numbers of gonorrhea cases precluded the intensive follow-up of each infected case that had been characteristic of the syphilis era.

In 1982 the World Health Organization/Pan American Health Organization (WHO/PAHO) identified the following key objectives for intervention to reduce STDs: (1) to minimize disease exposure by reducing sexual intercourse with persons who have a high probability of infection; (2) to prevent infection by increasing the use of condoms or other prophylactic barriers; (3) to detect and cure disease by implementing screening programs, providing effective diagnostic and treatment facilities, and promoting health-seeking behaviors; (4) to limit complications of infections by providing early treatment to symptomatic and asymptomatic infected individuals; and (5) to limit disease transmission within the community through the above efforts.

These objectives were used as a framework for the current United States program regarding STDs, which consists of the following components: (1) health education and promotion, (2) disease detection through testing and other means, (3) appropriate treatment, (4) contact-tracing and patient counseling, (5) clinical services, (6) training, and (7) research.

The Challenge of AIDS Control

It might seem reasonable to extend the experience in preventing the spread of other STD infections to AIDS. The objectives established by WHO/PAHO and the components of the current national STD program are certainly applicable to AIDS. However, AIDS presents a much different social problem than other STD infections. Since there is no cure for AIDS and no protection beyond avoiding or making safer intimate contact with infected individuals, those infected with the virus must be sexually isolated from uninfected persons. A condom barrier offers some but not complete protection. Avoidance of sexual contact and use of shared needles are the only sure protections.

Further, the stigma that accompanies a diagnosis of AIDS, based on fear and society's attitude toward IV drug abusers and homosexuals, presents a factor beyond the control of the infected individual or medicine. An HIV-seropositive individual who might live five years or much longer with no overt health problems, once identified in a community, may be subject to many and varied discriminations—by family and loved ones, by neighbors and friends, by employers and fellow employees, and by other providers of services.

As with prevention and control of all contagious diseases, prevention and control of AIDS involves two, sometimes competing, concerns. First, the person who is afflicted with the disease needs compassionate treatment, and neither those who have the disease nor those who have been infected with the virus should be subjected to irrational discrimination based on fear, prejudice or stereotype. Second, and of critical importance, the uninfected must be protected; those individuals who are not infected with the AIDS virus must have every opportunity to avoid transmission of the disease to them.

The Need for a National Policy on AIDS

Given the growing dimensions of the crisis and given limited national resources, it is imperative that a national policy be developed jointly by the public and private sectors. Such a policy must seek, in a cost-effective way, to achieve fundamental national goals: prevention, treatment, and cure—and adequate research in all three areas. A coherent national approach to this modern killer is needed: a comprehensive blueprint for a national response, not piecemeal solutions. Knowledge of the disease is now more than six years old and the growing magnitude of the problem has been apparent for nearly that long.

Such a national policy must have certain characteristics:

- The policy must be comprehensive, proceeding simultaneously on the fronts of prevention, treatment, and research.
- The policy must be coordinated between public and private sectors and between the different levels of government. A national policy does not necessarily mean a federal policy: there are important roles at all levels of the health care systems and at all levels of government. Nor does it necessarily mean uniformity: on certain issues different approaches should be tried to determine efficacy.
- The policy must be carefully balanced. For example, concern for the person with the disease must be balanced with concern for those who do not have the disease but who may become infected. Similarly, careful consideration must be given to directing scarce resources to increased prevention, even as increasingly large resources are necessarily devoted to research and treatment.
- The policy must be based on scientific information and medical judgments. Although policy choices must inevitably be made, they should be formed on the best available information and on the extensive public health experience in dealing both with AIDS and with other contagious diseases.
- The policy should be nonpartisan. Although it may be tempting to play on fears and prejudices, public figures and officials both inside and outside the health community should avoid exploiting the crisis for partisan political advantage.
- The policy should be capable of continuous review and modification as more and better information becomes available.

> *Recommendation 1:* A commission, modeled after the commission which made recommendations on the problems of Social Security financing in the early 1980s, should be constituted with representatives from the Executive branch of the federal government, the Congress, state and local government, and the private sector and di-

rected to develop a consensus position for consideration by the Congress, the Executive branch, state and local governments and private associations and institutions. The presidential commission announced, but not yet appointed, by the Administration could be broadened to implement this recommendation. A high-level body with representatives from the different branches and levels of government, but operating to the side of the more formal political processes, may have the best chance of forging the necessary national consensus which can then become the basis for concerted and coordinated action by both the public and private sectors.

The Special Role of Physicians and Other Health Care Counselors

Because there is no cure for AIDS, effective preventive techniques are vital. This involves both those who are infected and those who are not. Those who are infected must be identified so that they will not unknowingly transmit the disease to others. Many who are not infected will need to change their behavior substantially to minimize their risk of infection by the AIDS virus.

The key to changed behavior is public education coupled with counseling which must be given by physicians and other health care counselors.

Public Awareness

The public is well aware of AIDS in a general sense. The attention of the media has been intensively focused on the disease. Translating general awareness into modifications of behavior is the challenge.

The groups that are most at risk for AIDS, e.g., IV drug abusers, homosexuals, bisexuals, and prostitutes, have reason to know they are at risk. Their contacts, however, may not know they are at risk and hence spouses, unborn babies, and premarital and extramarital sexual partners may become infected. Education and counseling aimed at the high-risk groups must be the first priority. The education should urge immediate counseling with a physician or other health care counselor about the risk of AIDS, the uses of antibody testing and preventive measures.

Also, it must be recognized that persons in these groups may not respond to education and counseling and, when they do not, more aggressive programs—such as expanded methadone maintenance programs or penalties for knowingly exposing others—must be considered.

Education aimed at the more general population is difficult for at least two reasons. First, reaching all Americans with an effective message can be expensive and not all people respond in the same way or to the same method of learning. Messages must therefore be tailored to the target audience in question. Second, preventive messages must necessarily deal with controversial subject matter. Widespread use of the electronic media—especially television—appears to be the most effective way to reach the general public. Accordingly, public service advertising on the electronic media must be greatly increased, and these announcements must be shown at times and in places where they will be viewed by those who need the message most.

The AMA will continue its efforts to place its own public service ads on national television. AMA's Tony Danza public service advertisement (PSA) directed at teenagers about abstinence and condoms, and other PSAs which the networks have agreed to use, are significant first steps. But, more must be done and it must be nationally coordinated.

> *Recommendation 2:* The communications industry must develop voluntary guidelines for public service advertising regarding AIDS in consultation with the health care community and government officials. The AMA intends to be a catalyst in this effort to immediately bring the communications and health care communities together.

Counseling—And Educating Counselors

Perhaps the greatest need at the present time is effective counseling of both low-risk and high-risk populations by physicians or other health care counselors. A massive education effort for physicians and other counselors is necessary as a first step. Complete and accurate information on the disease, the modes of transmission, the appropriate application of antibody testing, and effective ways to change behavior must be understood by counselors if it is to be properly communicated to patients. In conjunction with face-to-face counseling, printed materials—like the Surgeon General's recent 36-page report on AIDS—should be widely disseminated.

Even more challenging than preparing physicians and others for generic counseling on AIDS is preparing these counselors to assist those who test positive and are infected with the virus. It is at that time that a change of behavior on the part of the person infected is most critical, and it is then that the most sophisticated counseling is required due to the emotional impact of the test results. There is no higher prevention priority than ensuring that the community of individuals who provide health care counseling be given adequate tools to be effective. And the AMA, as the largest organization of physicians in the world, must take a leading role in this undertaking.

> *Recommendation 3:* A conference should be immediately held between the AMA, other physician organizations and public health officials at all levels of government to determine: (1) the types of education and training that are necessary for effective counseling; (2) the people in the health care community who should receive this education and training; (3) the current resources available for such education and training; (4) recommendations for providing additional resources, including consideration of the respective roles of medical associations and government at all levels; (5) recommendations on how to update information continually as new scientific data are developed; and (6) recommendations as to alternative measures to prevent the spread of AIDS where education and counseling are not likely to be effective, particularly among IV drug users, through such programs as expanded methadone maintenance. The

AMA will promptly and widely report on the conference findings and assist in the implementation of the conference recommendations.

Voluntary and Mandatory Testing

Knowledge that a person is infected with the AIDS virus can be the crucial predicate to changing behavior. Thus, testing for an antibody to the AIDS virus, when used in conjunction with appropriate counseling (and when offered in the context of appropriate antidiscrimination and confidentiality protections discussed below), serves the important public health purpose of providing impetus for behavior changes that minimize the risk of transmitting the AIDS virus.

Clearly, the need for HIV-antibody testing has expanded beyond its original purpose, the screening of blood donors. Guidelines for the appropriate use of HIV-antibody testing must center on the following justifications: (1) to identify infected persons and to offer treatment where possible and to protect uninfected third parties, (2) to offer education and counseling that would modify high-risk behavior, (3) to solicit patient cooperation for locating and referring sex partners, and (4) to obtain broadened epidemiological statistics on the prevalence of HIV infection in the population.

In addition, in considering the merits of voluntary versus mandatory testing, these facts about AIDS must be kept in mind: (1) AIDS is caused by an infectious agent, and therefore is an infectious disease. Appropriate precautions, procedures, and policies should be applied to protect the community from the spread of the disease. (2) The extent to which the AIDS virus already has spread into the general population is not completely understood. Current projections are based on a number of unverified assumptions. (3) The transmission of the AIDS virus does not occur through casual contacts. Sexual contact, septic intravenous equipment, and the administration of infected blood and blood products are the main modes of transmission. (4) Heterosexual transmission of the AIDS virus, especially from males to females, does occur. (5) Seropositive pregnant females will transmit the virus to their babies in a high percentage of cases. (6) Health care workers, especially those who perform invasive surgical procedures, and emergency room and laboratory personnel, are at some risk when caring for AIDS patients. (7) No patient with a clinical case of AIDS has survived the disease. The disease has been uniformly fatal. (8) The disease, not its victims, is the threat from which society must be protected. (9) The confidentiality of the doctor-patient relationship is vitally important but not absolute. (10) Physicians have an ethical and professional obligation to behave in a scientifically responsible manner.

All of these considerations guided the Board of Trustees as it considered the issues that have been raised by the wide variety of proposals for HIV-antibody testing that are being discussed in society.

General Conclusions

Except for individuals in the limited categories listed in Recommendation 5 below (blood, organ and semen donors, immigrants, military personnel, prison inmates) with regard to whom testing serves well-established and well-accepted protection goals, mandatory national testing should not, at present, be broadly extended.

Military personnel have traditionally been subject to mandatory immunizations and our defense forces, of course, must be as strong as possible. Prison inmates, because they are confined and have a higher incidence of high-risk individuals than the general population, require special protection. Immigrants should be tested so that we can focus on the AIDS problem already here, and the nation certainly has the right to bar entrants with communicable diseases. The need to test donors of blood, organs and semen has never been questioned.

Public health authorities have advanced a plausible premise for their opposition to mandatory testing of homosexuals and drug abusers: such testing will only drive people underground and away from the health care system. Public health authorities also have advanced a premise for not requiring mandatory testing of large segments of the general population, such as all those seeking marriage licenses or all those admitted to hospitals: such testing in low prevalence populations would result in a high proportion of false positives, and would not be cost-effective, given the demand for voluntary testing and the shortage of testing and counseling resources for those who want them voluntarily or who will want them following effective public awareness campaigns.

Until those premises are shown by superior studies to be incorrect, a policy regarding mandatory testing which has been rejected by the vast majority of public health officials, including the Centers for Disease Control and the Surgeon General, cannot be recommended.

But certain high-risk groups should be regularly tested, with a right to informed consent and to refuse the test. Those groups are defined in Recommendation 6.

In addition, physicians and other hospital personnel involved in invasive surgical procedures who necessarily and unavoidably come in contact with the blood of patients, need to be aware of their risks. Limited regular testing of patients will assure that the CDC guidelines for the protection of hospital personnel are followed rigorously and will further assure that all patients receive prompt and full treatment. The Board emphasizes here that physicians have a long and honored tradition of tending to patients afflicted with infectious diseases with compassion and courage. That tradition must and will be continued throughout the AIDS epidemic.

Because the risk to health care personnel will be slight in most areas, any effort at mandatory testing of certain kinds of patients should be instituted after voluntary testing has failed and where a variety of factors, e.g., the costs and availability of proper testing and counseling as measured against the risk presented by the relative presence of a high-risk patient population, weigh in favor of mandatory testing.

The AMA does not believe it appropriate at this time to extend regularly offered testing to persons other than those listed, e.g., recommended testing should not be extended to all individuals anywhere who are considering marriage or to all persons in hospitals.

Decisions about whether there should be generally recommended testing to other types of individuals should, at this time, be left to the decision of the local community depending on its own circumstances and the judgments of its own public health officials.

At present, each case of AIDS must be reported by the individual physician to state public health authorities either by name or identifier. Anonymous, or if carefully implemented, confidential reporting should also be extended to all confirmed instances of persons infected with AIDS virus but not afflicted with AIDS Related Complex (ARC) or AIDS. Individuals who are seropositive for the HIV antibody are infected with the virus and can spread the disease as certainly as those with symptoms of AIDS. A sound epidemiologic understanding of the potential impact of AIDS on society requires the reporting of those who are confirmed as testing positive for the antibody to the AIDS virus.

Testing Recommendations

> *Recommendation 4:* Tests for the AIDS virus should be readily available to all who wish to be tested. The tests should be routinely subsidized for individuals who cannot afford to pay the cost of their test.
>
> *Recommendation 5:* Testing for the AIDS virus should be mandatory for donors of blood and blood fractions, organs and other tissues intended for transplantation in the United States or abroad, for donors of semen or ova collected for artificial insemination or in-vitro fertilization, for immigrants to the United States, for inmates in federal and state prisons and for military personnel.
>
> *Recommendation 6:* Voluntary testing should be regularly provided for the following types of individuals who give an informed consent: (1) patients at sexually transmitted disease clinics, (2) patients at drug abuse clinics, (3) pregnant women in high-risk areas in the first trimester of pregnancy, (4) individuals who are from areas with a high incidence of AIDS or who engage in high-risk behavior seeking family planning services, and (5) patients who are from areas with a high incidence of AIDS or who engage in high-risk behavior requiring surgical or other invasive procedures. If the voluntary policy is not sufficiently accepted, the hospital and medical staff should consider a mandatory program for the institution.
>
> *Recommendation 7:* As a matter of medical judgment, physicians should encourage voluntary HIV testing for individuals whose history or clinical status warrant this measure.
>
> *Recommendation 8:* Individuals who are found to be seropositive for the AIDS virus should be reported to appropriate public health officials on an anonymous or confidential basis with enough information to be epidemiologically significant.
>
> *Recommendation 9:* Physicians should counsel patients before tests for AIDS to educate them about effective behaviors to avoid the risk of AIDS for themselves and others. In public screening programs, counseling may be done in whatever form is appropriate given the resources and personnel available as long as effective counseling is provided.
>
> *Recommendation 10:* Physicians should counsel their patients who are found to be seropositive regarding (1) responsible behavior to prevent the spread of the disease, (2) strategies for health protection with a compromised immune system, and (3) the necessity of alerting sexual contacts, past (5 to 10 years) and present, regarding their possible infection by the AIDS virus. Long-term emotional support should be provided or arranged for seropositive individuals.
>
> *Recommendation 11:* Patients should knowingly and willingly give consent before a voluntary test is conducted.

Resources

Only recently has Congress and the Administration begun to seriously consider the vast resources needed to deal effectively with AIDS. Federal funding for 1988 is expected to reach $1 billion. But that amount will not be enough. The AMA endorses the bill introduced by Congressman Waxman to increase resources for testing and counseling.

Testing for the HIV virus in America will require substantially more resources than are currently being made available. Trained counselors, materials for counseling, and research on effective counseling approaches, for the variety of population groups that need these services, are urgently required. Also, dependable testing facilities with sufficient capacity to respond to the epidemic are needed now. In addition, funds for research and care must be increased to fully exploit the nation's capacity to respond effectively to this crisis.

The key premise of a prevention strategy, when there is no vaccine, is behavioral change on the part of those infected and those at risk of infection by AIDS virus. It is therefore crucial that there be immediate and systematic studies conducted of how behavior of affected groups may have changed in recent years, and if possible, what factors caused the changes. Most particularly, it is necessary to study and evaluate the types of counseling that have been effective so that the techniques may be replicated widely. There can be little question that in a free society suasion and voluntary change, if effective, are far preferable to compulsion.

> *Recommendation 12:* Public funding must be provided in an amount sufficient (1) to promptly and efficiently counsel and test for AIDS, (2) to conduct the research necessary to find a cure and develop an effective vaccine, (3) to perform studies to evaluate the efficiency of counseling and education programs on changing behavior, and (4) to assist in the care of AIDS patients who cannot afford proper care or who cannot find appropriate facilities for treatment and care.

Protection Against Discrimination

Anti-Discrimination

The AMA believes strongly that AIDS victims and those who test positively for the antibody to the AIDS virus should not be treated unfairly or suffer from arbitrary or irrational discrimination in their daily lives. Last year, the AMA filed a friend of the court brief in *School Board of Nassau County v. Arline,* a case before the Supreme Court which addressed the question of how the federal handicapped anti-discrimination laws should apply to persons afflicted with contagious diseases. The AMA set forth a framework for the application of the law which the Supreme Court adopted, quoting verbatim from the AMA brief in its key holding.

A sound anti-discrimination approach does not allow reflexive discrimination against AIDS victims based on fear or stereotype or prejudice. Nor does it require that all employers or other federal fund recipients automatically accommodate a person afflicted with a communicable disease. Instead, based on an individualized analysis of the nature and duration of the handicap and the nature and duration of the communicability, a federal fund recipient must make a reasonable accommodation based on reasonable medical judgments, given the state of medical knowledge at the time. This sound framework for carefully balancing the two competing concerns—the right of the victim to be free from irrational acts of prejudice and the right of others to be protected against an unreasonable risk from disease—should also guide state anti-discrimination efforts.

A key question left open by the Supreme Court is whether a person who is not afflicted with AIDS or ARC, but who nonetheless tests positive for the antibody, is protected by the federal anti-discrimination law.

In order to encourage people to seek counseling, and testing if necessary, the AMA strongly urges that anti-discrimination laws at both the federal and state levels be clarified either by regulatory interpretation or statutory amendment to cover those who test HIV antibody positive. Allowing irrational discrimination against those who test positive serves no useful purpose: it only has the destructive effect of removing those who are otherwise productive members of society from the work force or otherwise denying them access to an important aspect of normal life. While the federal law should continue to apply only to federal fund recipients, state laws should be sought to prevent irrational discrimination by entities or individuals within those jurisdictions.

> *Recommendation 13:* Anti-discrimination laws must be clarified or amended to cover those who test positive for the antibodies to the AIDS virus.

Confidentiality

The ability of the health care community to maintain the confidentiality of patient information and restrict its use to only those purposes essential for maintenance of health is, like clarification of anti-discrimination laws, vital to an effective program of preventing and controlling AIDS. Even if anti-discrimination laws were completely effective, which unfortunately is not likely, persons who test positive (such as those with ARC or AIDS), will suffer stigma. Thus, confidentiality is crucial.

The basic principle should be that access to patient information should be limited only to health care personnel who have a legitimate need to have access to the information in order to assist the patient or to protect the health of others closely associated with the patient.

As with anti-discrimination laws, laws protecting the confidentiality of patient information should be on both federal and state agendas.

> *Recommendation 14:* Model confidentiality laws must be drafted which can be adopted at all levels of government to encourage as much uniformity as possible in protecting the identity of AIDS patients and carriers, except where the public health requires otherwise.

Questions for the Future

As the national debate on prevention and control of AIDS continues, other important issues will need to be addressed.

Research and Data

There is an urgent and critical need for more scientifically sound data on the prevalence and spread of virus in the general population. At the present time only those cases that meet the current CDC surveillance definition of AIDS are reported to that institution. Since AIDS is the terminal and fatal stage of HIV infection, it represents only the tip of the huge HIV-infection iceberg. There are protean manifestations of HIV infection ranging from infected asymptomatic to full-blown AIDS. How large the base of that iceberg really is—that is, how many people are actually infected—can only be estimated from the number of reported AIDS cases. That has been done by using a multiple (50 to 100 times the number of AIDS cases) that has been extracted largely from surveys done in high-prevalence areas. Yet this same multiple has been used to estimate the number of current and potential HIV-infected persons in low-prevalence areas and for that matter the entire country and even the world. The CDC itself is unsure about the accuracy of its estimates. Yet if economic and medical plans are to be made for the future, reliable projections must be available. How sufficient or exaggerated these plans may be depends upon the accuracy of current and future estimates of HIV-infected persons, particularly as to the extent of its spread into the low-risk heterosexual population.

Not only are accurate estimates of HIV-infected persons needed, but so too are reliable data on the rate conversion of asymptomatic seropositive persons to clinical illness, including AIDS, that requires increased medical care. This information is important for the formulation of plans for the future cases of potentially hospitalizable patients and the economic consideration thereof. HIV infection has protean manifestations and death can result not only from AIDS itself, but from severe ARC or progressive CNS disease as well. In order to obtain accurate information in HIV-infected persons on the rate of conversion from asymptomatic to clinically severe illness, baseline data on their serologic status must be obtained as early as possible—not after clinically manifest disease is present. The presence of HIV antibodies indicates not only current infection with the virus, but also that the patient is potentially capable of transmitting the disease. This follows from the fact that HIV integrates its genome into the host cell genome with the result that once infected, the patient remains infected for life and is, therefore, capable of lifelong transmission of the agent. The earlier the infected person is detected, the earlier he or she may be advised of this contagious state and counseled on how to avoid further transmission of this lethal virus.

> *Recommendation 15:* Consistent with the proposal by the Secretary of Health and Human Services, a national study in various areas of the country must be immediately undertaken to determine the prevalence and conversion rate of the virus in the United States population, and the study must be repeated at appropriate intervals to gauge the spread of the disease.

Warning to Third Parties

One of the more difficult issues for society is how to warn unsuspecting spouses or sexual partners of persons who test HIV positive. Such a warning would allow the third party to practice "safer" sex or to abstain from sexual relations with the infected person altogether. Given the life-or-death consequences, the unsuspecting third party should, as a general matter, be warned because there is no cure and because it may not be responsible to rely solely on the infected person to provide a suitable warning.

Physicians who have reason to believe that there is an unsuspecting sexual partner of an infected individual should be encouraged to inform public health authorities. The duty to warn the unsuspecting sexual partner should then reside in the public health authorities as well as the infected person and not in the physician to the infected person.

The AMA believes that mechanisms, analogous to those used by public health authorities to warn sexual partners about other STDs, should be put in place to warn unsuspecting third parties about an infected sexual partner. Such warning may be appropriate whether the infected person is bisexual, heterosexual or homosexual.

This problem raises the general question of whether anonymous reporting should continue to be the standard for persons who test seropositive. Our recommendation at this time is limited to situations where physicians or health officials already know the identity of the AIDS carrier and have reason to believe a risk to third parties exists.

> *Recommendation 16:* Specific statutes must be drafted which, while protecting to the greatest extent possible the confidentiality of patient information (1) provide a method for warning unsuspecting sexual partners, (2) protect physicians from liability for failure to warn the unsuspecting third party but (3) establish clear standards for when a physician should inform the public health authorities, and (4) provide clear guidelines for public health authorities who need to trace the unsuspecting sexual partners of the infected person.

Sanctions for Reckless Disregard for the Safety of Others

A related question which must be explored is whether an infected person, who knows he or she is infected and who knowingly fails to warn a sexual partner of the infection, should be subject not just to tort suits, but to a proceeding brought by state authorities to sanction the individual.

> *Recommendation 17:* Given the risk of infection being transmitted sexually, and given the dire potential consequences of transmission, serious consideration should be given to sanctions, at least in circumstances where an unsuspecting sexual partner subsequently finds out about a partner's infection and brings a complaint to the attention of authorities. Preemptive sanctions are not being endorsed by this recommendation.

Conclusion

The Board intends to review its evaluation of the developing AIDS epidemic on a constant basis. Modifications of the AMA's positions will be made as the situation warrants.

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Renal Hypertension Following Conservative Management of Blunt Renal Trauma

FREDERICK A. KLEIN, M.D. and LOUIS E. ELKINS, M.D.

Introduction

Although considerable controversy existed five to ten years ago regarding conservative versus early operative management for blunt renal trauma, most urologists managing renal injuries today reserve early exploration for those patients with renal pedicle injuries, "shattered" kidneys or patients who are hemodynamically unstable. The long-term complications of conservative management are few, but do include hypertension secondary to a "Page" or "Goldblatt" kidney, arteriovenous malformations, renal artery aneurysms, urinoma and/or abscess formation. Because of these possible complications, compulsive periodic follow-up evaluation should be performed in these patients. Herein we describe a multiple trauma patient who suffered a class 2 renal injury that was treated conservatively, developed renovascular hypertension with lateralizing renin elevations at four months after injury, and was subsequently managed successfully by heminephrectomy.

Case Report

An 18-year-old previously healthy white man was involved in a single car accident at 70 mph while racing his stock car at a local track. Initial resuscitation was accomplished at an outlying hospital where the patient's blood pressure was stable at 130/80 mm Hg; he was unconscious, with small pupils minimally reactive to light and urine that was grossly bloody after a Foley catheter was inserted. He was transferred via helicopter to the University of Tennessee Medical Center in Knoxville where his trauma score was 16 and his injury severity score was 38. He was arousable, responsive to commands, and able to move all extremities. There were abrasions on the right arm, chest, and leg. His right chest, abdomen and right costovertebral angle were tender to palpation, and his urine was grossly bloody; his blood pressure stable at 140/90 mm Hg. The initial CBC count revealed a WBC count of 22,700/cu mm, hemoglobin 14.1 gm/dl, and hematocrit of 40.8%. Electrolytes were normal. Subsequent evaluation revealed fractures of the right 9th, 10th, and 11th ribs with a pulmonary contusion on chest x-ray. A computed tomography (CT) scan of the head was normal; scan of the neck revealed a non-displaced fracture of C5, and a scan of the abdomen and retroperitoneum revealed a severe class 2 cortico-medullary laceration with minimal urinary extravasation (Figs. 1 and 2). There was no evidence of other intra-abdominal injury. He was admitted to the intensive care unit where over the next 14 days his mental status returned to

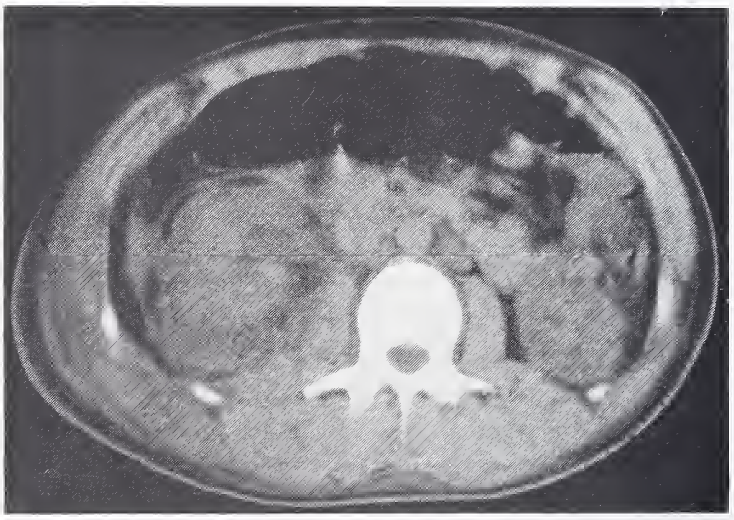

Figure 1. CT scan through abdomen without contrast showing large right perirenal hematoma and ruptured kidney.

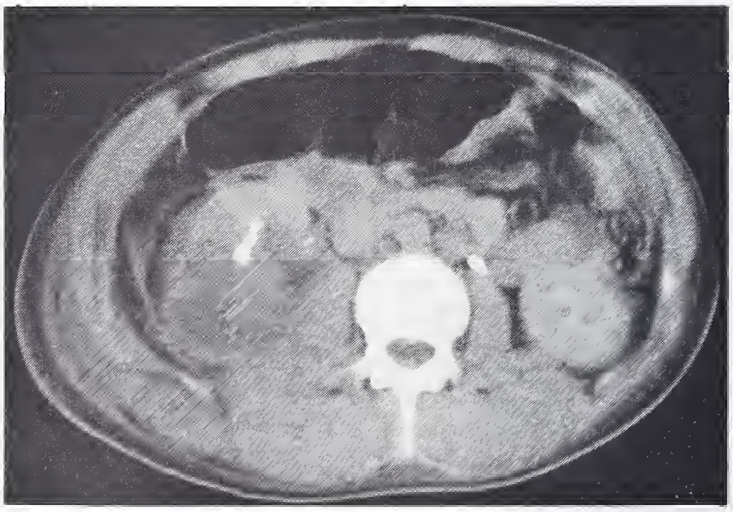

Figure 2. CT scan through abdomen with contrast showing non-functioning lower pole and perirenal hematoma.

From the Division of Urology, Department of Surgery, University of Tennessee Medical Center at Knoxville.

baseline and his hematuria cleared. Although he had a right lower lobe pneumonia and a hemoglobin drop to 9.2 gm/dl, his blood pressure remained stable and he was discharged on the 17th day following admission, for follow-up in one month.

Subsequent examinations revealed good healing of his neck and rib fractures and a return of his hemoglobin to baseline levels. Blood pressure readings, however, remained consistently around 160-170/100-110 mm Hg for three months. This prompted readmission to the hospital, where renal arteriography and renin values were obtained. The arteriogram (Fig. 3) revealed a right lower pole scar due to a segmental infarct. The renin values were significantly elevated on the right, with a value of 4.3 ng/ml compared to 1.5 ng/ml on the left and 1.1 ng/ml in the inferior vena cava. Because of renovascular hypertension, a right partial nephrectomy was performed four months after injury. Six weeks later a CT scan (Fig. 4) revealed a normally functioning upper pole segment. His blood pressure was 110/70 mm Hg postoperatively, and at follow-up three months after surgery, his blood pressure remains 110/70 mm Hg and he has resumed his regular occupation.

Discussion

Renal injury accounts for approximately 8% to 10% of patients seen for abdominal trauma secondary to blunt or penetrating injuries.[1] Blunt trauma accounts for about 80% of renal injuries, most commonly the result of a motor vehicle accident, fall, contact sports, or fights. The mechanism of injury may be rapid deceleration, causing excessive movement of the kidney, with stretching of the vessels, which in turn leads to major vascular trauma, or by means of a direct blow, where the kidney may internally be thrust against the rib cage or vertebrae, or externally against a solid object resulting in contusion, laceration, or avulsion of renal parenchyma.[1]

The single most common sign suggesting renal injury is hematuria, some degree of which occurs in well over 90% of cases in most series. It should be emphasized, however, that the degree of hematuria does not necessarily correlate with the severity of the renal injury. Glenn and Harvard[2] reported that in 44 patients with gross hematuria, 17 had insignificant renal injury. On the other hand, numerous authors have reported major renal injuries to occur with little or no microscopic hematuria.[1] Such cases, usually with severe injuries in which the renal pedicle is either avulsed or thrombosed, are nearly always associated with other organ injuries (splenic 57%, hepatic 40%), and can usually be diagnosed because of a high index of suspicion.[1]

The most commonly used nomenclature divides renal trauma into three separate groups: minor parenchymal laceration or contusion, major parenchymal laceration through the corticomedullary junction and often involving the collecting system, and renal pedicle injury or a "shattered" kidney.[3] In the 1977 Parkland Hospital blunt trauma series, which included 115 patients admitted for treatment, there were 54 class 1 injuries (47%), 36 class 2 injuries (31%), and 25 class 3 injuries (22%).[4] In a more recent series of 185 consecutive renal injuries requiring surgery, renal pedicle damage from blunt trauma accounted for 14% of the cases seen.[5]

The evaluation of renal injuries should be done in a systematic fashion, and includes a careful history, physical examination, and urinalysis. Whether the initial study for renal evaluation should be an intravenous pyelogram or CT scan

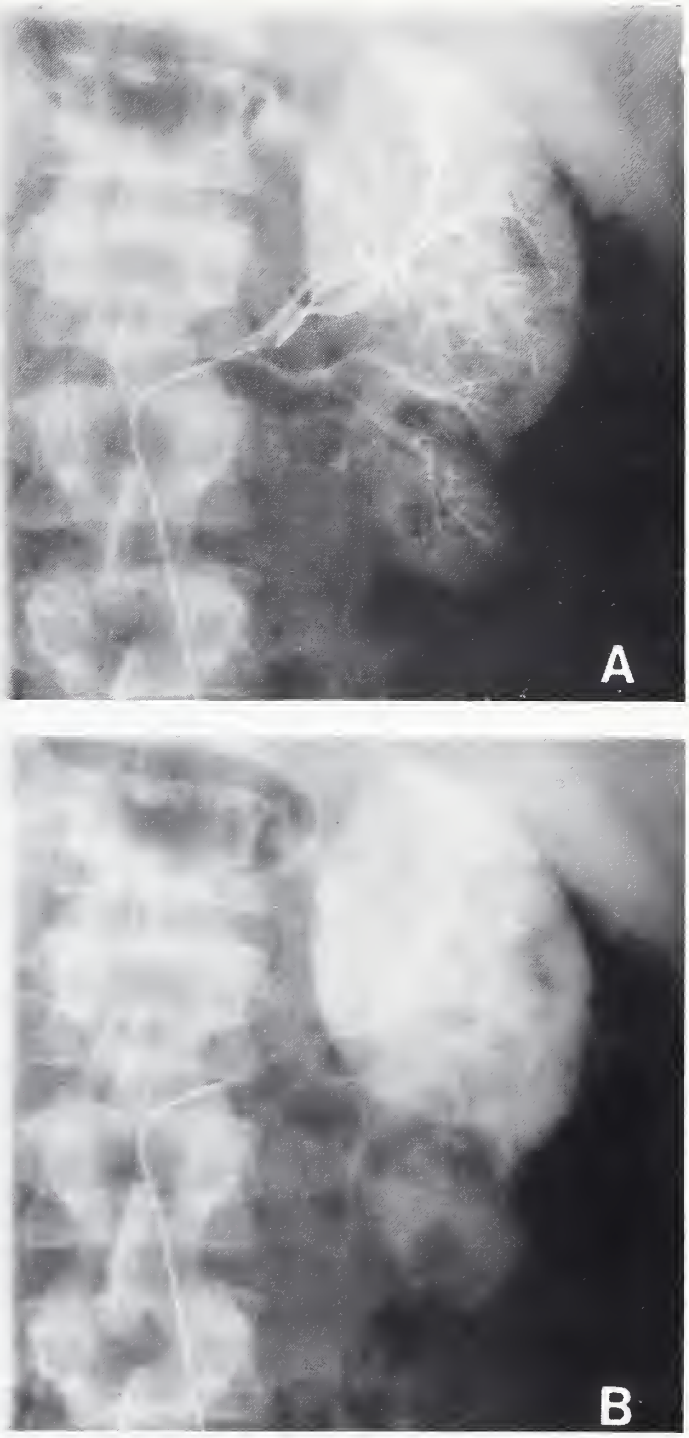

Figure 3. Selective right renal angiogram arterial (A) and venous (B) phases revealing segmental scarring and poor perfusion of lower pole.

mainly depends upon the availability of a CT unit for emergency studies and the preference of the treating physician. There is little doubt that CT scans have several advantages over pyelography or arteriography, which include three-dimensional views, non-invasiveness, excellent definition of parenchymal lacerations, clear demonstration of extravasation, precise outline of non-viable tissue, definition of extent and size of surrounding hematomas, and detection of associated organ injury.[1] Since it is now pretty much the standard of care to use CT to evaluate the abdomen after blunt trauma, concomitant evaluation of the kidneys and retroperitoneum are obtained, allowing one to determine what type of management is necessary.

The decision for surgical or medical management of a patient with blunt renal trauma should be based on the clinical status of the patient and the previously mentioned classification. The decision should not be made without proper radiologic definition of the injury. Once the extent of the injury is defined, then treatment may be selected, based objectively on expected eventual complications. Eighty-five to ninety percent of blunt renal injuries are minor, with renal contusion or superficial cortical laceration. These injuries are uniformly managed nonoperatively, with few serious sequelae; they do not lead to hypertension, abscess, loss of renal function, or delayed bleeding. Although there may be associated injuries, they are much less common than in the patients with a more severely damaged kidney.[1,6]

Ten to fifteen percent of patients who suffer blunt renal trauma suffer major injuries,[6] such as lacerations through the cortex and medulla without extravasation, laceration through the cortex and medulla with extravasation, and vascular injuries. Vascular injuries obviously need surgical correction as quickly as possible, though there is controversy as to whether early exploration or conservative mangement is appropriate when a deep laceration occurs with or without extravasation. The primary difficulty lies in determining which patient will not suffer complications with nonoperative management. The specific indications for renal exploration should include expanding or uncontained hematoma, pulsatile hematoma, moderate to severe urinary extravasation, nonviable renal parenchyma, and vascular injury. Though major cortical lacerations and shattered kidneys with fragments contained within Gerota's fascia will heal, delayed bleeding and hypertension may occur,[6] and when managed conservatively, such patients should stay at bed rest from 10 to 14 days and have periodic long-term follow-up. Using these criteria and the important surgical principle of midline approach with early control of the renal vessels when exploration is performed, the incidence of nephrectomy is less than 10% and secondary nephrectomy around 2%.[6]

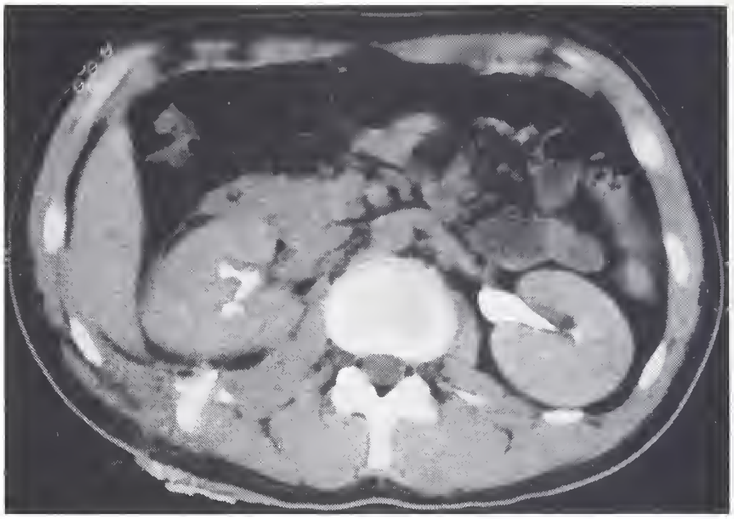
Figure 4. Postoperative CT scan showing good function of remaining upper pole segment.

Early complications of conservatively managed renal trauma include delayed bleeding, abscess formation, urine extravasation, sepsis, fistula formation, and hypertension. Late complications include arteriovenous fistula formation, hydronephrosis, calculus formation, chronic pyelonephritis, pain, and persistent hypertension. Fortunately, most of these complications, including significant hypertension, are unusual.

The exact incidence of hypertension following renal trauma is unknown, but it is relatively small.[7] In most instances it is transient, and usually resolves within the first six weeks after injury, but it may occur as late as ten years or longer after injury. Since Goldblatt[8] in 1934 demonstrated that renal artery stenosis could produce hypertension in dogs, and Page[9] showed in 1939 that cellophane wrapped kidneys produced hypertension in dogs, renovascular hypertension in man has been recognized. Braasch and Strom[10] reported in 1943 that three of 50 patients who suffered renal trauma developed hypertension, which was subsequently relieved by nephrectomy, and in 1955 Engel and Page[11] reported the case of a 19-year-old man with hypertension developing from a subcapsular hematoma. Glenn and Harvard[2] reported hypertension in five of 84 patients following renal trauma, and eight similar cases of hypertension from subcapsular hematomas were reported by Downs and

Hewett in 1962.[12] Since then, this phenomenon has been well recognized and reported by others.

The mechanisms of posttraumatic renal hypertension include parenchymal compression from hematomas (Page kidneys), and renal artery stenosis or segmental arterial occlusion, analogous to the Goldblatt kidney. Because of the possibility of hypertension, patients with significant renal trauma need to have periodic follow-up. If hypertension is found, appropriate evaluation should be undertaken; this should include arteriography and renin sampling. As in our case, where a segmental infarct and lateralizing elevated renin values were found, the responsible lesion should be readily recognized. Treatment then consists of either renal artery repair, nephrectomy, or partial nephrectomy as the identifiable lesion dictates. With appropriate management, most patients' hypertension can be controlled.

The current trend in managing blunt renal injuries is toward conservatism unless there is hemodynamic instability or pedicle injury. It should be emphasized, however, that since delayed hypertension may develop, patients treated medically require periodic follow-up. If delayed problems do occur, renal preserving procedures can often be performed; on the other hand, nephrectomy is more often the result if there is immediate exploration.

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Health Risk Factors of Tennessee's Teenagers

ALLEN BOLTON and EVELYN ROBERTS

For several years the Health Promotion Section of the Tennessee Department of Health and Environment, in an effort to effectively direct health education, has sought valid information on the health-related beliefs and behaviors of teenagers in the state. National school health education evaluation specialists have also expressed the need for further research to validate the health practices of teenagers.[1] In June 1986, the Teen Wellness Check (TWC) Research Project was commissioned to accomplish this task in Tennessee.

During the 1986/1987 school year, a representative study of Tennessee's 9th and 12th graders was conducted. The University of Tennessee Department of Health, Leisure, and Safety assisted with study design, data collection, and analysis. A computerized health risk appraisal, the Rhode Island Teen Wellness Check, was used to collect information from students in the classroom. The

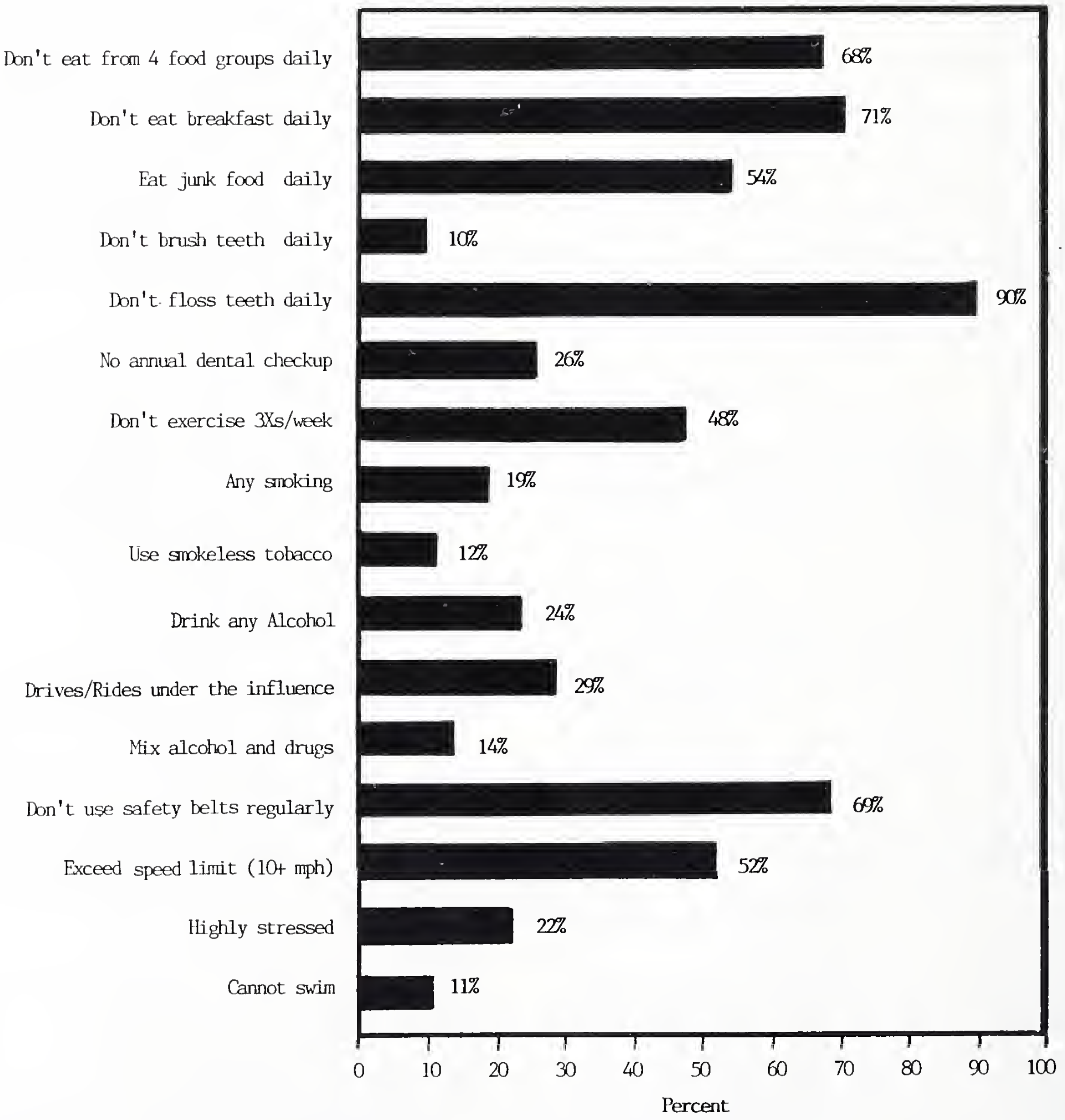

Figure 1. Health risk factors of Tennessee 9th and 12th graders.

From the Tennessee Department of Health and Environment, Nashville.

data were aggregated to insure confidentiality of the students and to provide a file for composite analysis. Seven schools were selected and 1,506 students surveyed. Fig. 1 provides an overview of the prevalence rates for the particular behaviors that were assessed. Analysis of the data by demographic variables indicated differences in rates between and among different groups. The topics of greatest concern to the Health Promotion Section are highway safety, nutrition, fitness, and tobacco use.

Rates for highway safety practices are perhaps the most troublesome because they can have the greatest impact upon the immediate health status of teenagers. The majority of 9th and 12th graders reported not wearing seatbelts regularly. More than one-half of the respondents admitted to exceeding the speed limit by 10 miles per hour or more, and although not legally permitted to drink, one out of every three told of driving or riding with a driver under the influence of alcohol. These risk factors and the inexperience of most teenagers as motor vehicle operators make motor vehicle accidents the leading cause of death for persons 15 to 19 years of age.[2]

Proper nutrition practices were reported infrequently, with one-half to three-fourths of respondents indicating less than adequate nutritional habits. Among teenagers, diets consisting mainly of nutrient-sparse foods are more typical than diets including the four basic food groups. Specifically, they do not eat from the four basic food groups daily, do not typically eat breakfast, and regularly eat "junk" food.

Recent national data indicate that adult Tennesseans are among the most physically inactive people in the United States.[3] TWC data suggest this trend toward sedentary lifestyles begins at an early age. One-half of the respondents admittedly did not employ "adequate" personal fitness practices, defined as the minimal level of aerobic activity required to achieve cardiovascular benefit, e.g., nonstop aerobic activity for 20 or more minutes, three or more times per week.

By the ninth grade, prevalence rates for tobacco use have been established, indicating a young age for initiation of this behavior. During the past decade, various studies have documented that the number of female smokers has increased and their rate, as indicated by TWC data, is equal to the smoking rate for men. A more detailed analysis of the TWC data demonstrated that the men who now use smokeless tobacco outnumber those who smoke (Fig. 2). Many declared that they believe smokeless tobacco to be a safe alternative to cigarettes.

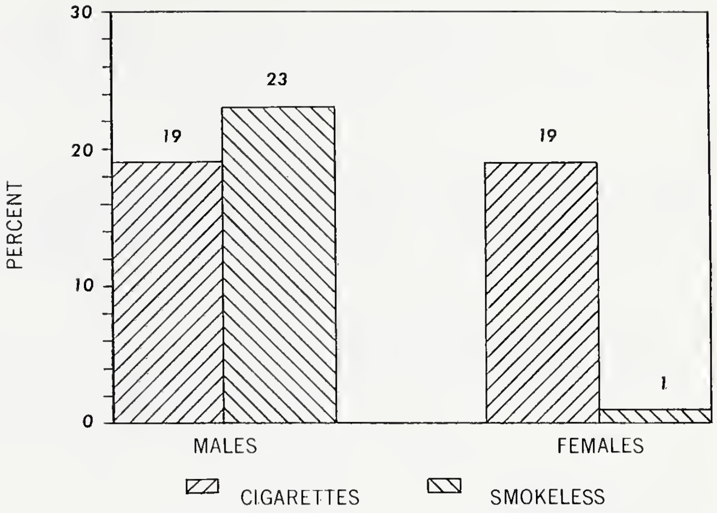

Figure 2. Adolescent tobacco use.

The real benefit of research into the identification of health risk factors of individuals is the stimulation of actions taken to prevent or reduce those risk factors. The data presented, while not necessarily conclusive, are at least directive. Several interesting and troubling facts have surfaced; some document previously held notions while other commonly held beliefs are disproved. In either case, the need for corrective action is clearly demonstrated. The Tennessee Department of Health and Environment, through its statewide Health Promotion and School Health Programs, will be implementing projects in some secondary schools that attempt to reduce the risk behavior of the state's adolescent population. These projects will target specific health risk factors identified by the TWC Research Project. The Department will also be working with other departments and agencies, interpreting the data and assisting in the design of appropriate courses of action. Other health and education professionals are encouraged to utilize the TWC information to direct health promotion efforts with children and adolescents.

For further information about the TWC Research Project or for a copy of the complete report, call or write: TWC, Health Promotion Section, 100 9th Ave. North, Nashville, TN 37219-5405, Tel. (615) 741-7366.

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Loss Prevention Case of the Month

Excellent Care—Unexpected Result

J. KELLEY AVERY, M.D.

Case Report

In a single car, high-speed accident a 16-year-old boy sustained multiple contusions and fractures, including a fracture of the dorsal spine with significant displacement, which required emergency surgery to relieve and/or prevent pressure on the spinal cord. A laminectomy was done at T-10 within the first 12 hours of admission and the patient stabilized uneventfully. Within the first 48 hours after his surgery, some loss of upward gaze on the left was noted, but he denied diplopia and visual acuity seemed unaffected; on examination, however, there was some inability of the left eye to move upward with the right eye. The neurosurgeon who had attended the patient initially called in an excellent plastic surgeon with extensive maxillofacial experience for consultation. Additional x-rays of his face revealed a fracture of the floor of the orbit on the left (a blow-out fracture), and surgery was planned to relieve what was felt to be some entrapment of the external ocular muscles. Surgery was carried out uneventfully with the reduction of the fracture of the floor of the left orbit and no apparent difficulties were noted during the operation, but upon reacting the patient complained of loss of vision on the left; it never returned.

A lawsuit was filed, charging the plastic and reconstructive surgeon with negligence in the performance of the operation, as well as lack of informed consent. As usual, depositions followed with expert witnesses being summoned by both sides. Though absolutely no justification was found for the charge of negligence related to the surgical procedure, numerous expert witnesses were available to testify that failure to inform the patient and/or the parents of this 16-year-old boy of the possibility of the loss of vision as a complication of the surgery was a deviation from an acceptable standard. A trial followed and there was a large jury award based solely on the lack of informed consent.

Loss Prevention Comments

More and more lawsuits are being brought and more and more money is being paid out because, after a bad result, the plaintiff patient believes that he was poorly informed as to the possible consequences of a surgical procedure. In this case, this young man had excellent care from both his neurosurgeon and his plastic and reconstructive surgeon. The complication that occurred, causing him to lose the vision in his left eye, is extremely serious and extremely regrettable, but it nevertheless can and does occur in this kind of surgery, even when done by experts in the field.

The question normally is raised, "How much documentation is necessary to avoid this kind of lawsuit?" Our claims attorneys at SVMIC say that cases of this type where settlements are required or awards are given are those cases where there is no evidence in the record of any encounter between the patient and the operating surgeon during which an attempt is made to apprise the patient and/or family of the potential risks and benefits of the planned surgery. As was true in this case, the record is simply silent on the entire subject. The surgeon could not specifically remember warning of the possibility of blindness, however unlikely, as a complication of the surgery, and of course the parents stated that there was no mention of such a complication. It is worth emphasizing that SVMIC has yet to lose or settle a case because of lack of informed consent where there is a note by the attending physician that he talked with the patient and discussed the risks, and the patient seemed to understand the essence of the discussion. Any discussion should include any extremely serious complication that can occur, i.e., death, blindness, paralysis, loss of limb, so as to provide the patient with all of the knowledge that he needs to make an informed decision relative to consenting to allow surgery.

Patients have been conditioned to expect unrealistically good results from our efforts, and because of this, when an unexpected or bad result occurs, patients will always believe that they should have known about its possibility before the fact and, on occasion, this dissatisfaction will result in the filing of a lawsuit.

Dr. Avery is chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood, and medical director for Ambulatory/Outpatient Services, St. Thomas Hospital, Nashville.

James T. Galyon

Tort Reform

Very little is being said in the Tennessee news media about the tort system and its reform. There was a great deal of activity last year in forming a coalition for civil justice in an effort to consolidate the interest of the varied and many parties seeking relief from the injustices of the current tort system.

The Governor had appointed a special task force on tort reform. It heard testimony all around the state and reached conclusions from which many useful recommendations were made, but the legislature failed to act on these recommendations. When the state legislature reconvenes in January, one of our prime concerns should be persistence in trying to obtain changes in the tort system.

Several problems are evident in the current system. First, there is a steady movement toward no fault liability which increasingly finds individuals liable for damages even if no wrongdoing can be found on their part.

Second, the plaintiffs' attorneys have developed a variety of questionable tactics and doctrines that shift the liability to those with so-called deep pockets, even though they did not cause the damages or had a very limited role in causing the damages. This is particularly reprehensible when the injured party is to a large extent responsible for his own damages.

Third, there has literally been an explosion of unrealistic awards for damages, particularly for non-economic damages such as pain and suffering or punitive damages.

Fourth, the entire system is inefficient and unfair to all parties, particularly the truly injured parties who should receive compensatory damages. Currently two-thirds of the dollar cost of the system is absorbed by attorneys and court costs of this legal system.

In the medical field the costs are even greater. The direct costs are ever-increasing malpractice premiums which must be passed on to the patient. The indirect costs are even greater, such as the widespread practice of defensive medicine and the hidden cost to defend frivolous lawsuits. In 1984 the direct cost of malpractice insurance and lawsuits was $60.5 billion or 16.8% of the health care cost of the nation. This translates to 1.76% of the GNP. This is particularly wasteful considering that only 30% to 40% ever reaches the injured parties.

Medicine is reacting to this in a predictable manner—high-risk specialties are withdrawing their services. The availability of obstetrical care is the most obvious of these services now being withdrawn in Tennessee. There are at least 12 counties in which no physician offers obstetrical deliveries. There is no reason to think that we will be spared the withdrawal of other specialty services such as neurosurgery, orthopedics, etc. We can now see this happening in Florida and elsewhere.

The problem seems to be apparent to all except the trial lawyers and certain of the news media. What are the remedies? For starters, I have the following recommendations: (1) Return to a true fault based liability, based on scientifically credible evidence and opinions. (2) Eliminate joint and several liability discouraging the deep pocket enticement. (3) Limit damages, particularly non-economic losses such as pain and suffering, to a reasonable amount. (4) Provide structured periodic payments rather than lump sum, front-end payments for large awards. (5) Reduce the inefficiency of the system by limiting attorneys' contingency fees to a reasonable amount based on a sliding scale. Ultimately new alternatives to resolve these disputes out of the court system must be developed.

James T. Galyon, M.D.

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SEPTEMBER, 1987

editorials

Congratulations!

When I became editor of the *Journal* in January 1972, I had had a six-month apprenticeship under the previous editor, Rudolph Kampmeier, M.D., but you couldn't say that at the end of that time I knew much at all about being an editor. Though I turned often to Rudie, and to Ad Scoville, who was then, and still is, the associate editor, the nuts and bolts and the day-to-day joys and trials of editing I learned from the assistant executive director of the Association, Mr. Hadley Williams, who had then been with the Association for nearly ten years. I owe him a lot.

When Jack Ballentine retired as executive director of the Association in 1976, it was only natural that Hadley would be named to succeed him. The magnitude of the job and its tribulations have compounded in the past few years, as medicine has ceased being a sort of club and pretty much the private domain of doctors, and became everybody's business—sometimes it seems the doctors' least of all—in a litigious society that wants to say not only how it should be treated, but also afterwards how it has been treated; in all of this it is undeterred by ignorance. During his tenure, Hadley has served us well, and the Association has prospered under his direction, especially considering the inherent hazards.

The American Association of Medical Society Executives is to the execs what the AMA is to doctors, except that its membership is close to 100%, and includes executives in both state and local as well as national medical societies. Its primary function is to help them help us to deliver health care more effectively to the people of this country. Recognition in that organization came to Hadley in 1981 with his election to its Board of Directors. To those of us who know him well and had worked closely with him it seemed only natural that he would go to the top, and he did.

On July 31, 1987, Mr. L. Hadley Williams, executive director of the Tennessee Medical Association, was installed as president of the American Association of Medical Society Executives. The *Journal* extends congratulations and best wishes to him, knowing that he can fill that position with distinction while at the same time still serving TMA in the exemplary manner he always has.

J.B.T.

"Big Daddy" Revisited

Like the republic at large, American Medicine (used here as the doctors who comprise it) is divided, and along much the same lines. We are multi-racial, we are black and white, Protestant, Catholic, and Jew, and none of the above. Some of us are "pro-life" (usually more properly anti-abortion), and those who are would say some of us are pro-death, by which they mean pro-abortion and in favor of the right to die; some of us are split as well on those two. Some of us—most of us—are in private practice, being compensated by fee for service as much as we can; more and more of us, though, are employed physicians. Some of us are Republicans, some are Democrats, some are liberal and some conservative, but more often it is true that we are liberal on some things and conservative on others—without consensus.

On the other hand, American Medicine is not divided at all, except as the republic itself is divided. If you want to see how we are not divided, I commend to you the most excellent inaugural address of the distinguished President of the American Medical Association, William S. Hotchkiss, M.D., published elswhere in this issue of the *Journal.* Our mission is to care for the sick and to prevent sickness in the world. It is to be advocates for our patients when corporate or governmental agencies would put fiscal considerations before their welfare. It is to combat mediocrity, and the forces within and without the profession that encourage it.

Neither the Democratic nor the Republican party—no, nor any of its candidates—can speak for the individual citizen of the United States in every situation, even though that is what we elect them to do. It is that expectation that accounts for the perpetually renewed composition of the Congress. It is equally unrealistic, when we can have within our own ranks such disparate organizations as, for example, the Physicians for Social Responsibility and the Association of American Physicians and Surgeons, and dedicated active workers in both the Republican and Democratic parties, to expect that American Medicine could have an umbrella organization that could speak for it on socioeconomic-political matters, yet that is the basis on which some American doctors eschew membership in the AMA.

That sort of thing is not what the AMA was formed to do. It is true that much AMA staff effort goes into legislative activity, but detractors of the AMA wrongly attribute that to attempts at feathering its members' nest. In fact, most of those efforts are aimed at protecting our patients from intrusion into the doctor-patient relationship that would result in inferior patient care or diminished services. We are, as Dr. Hotchkiss points out, a service organization—for service to our membership, to be sure, but that role is relatively minor. The AMA is first of all a *public* service organization. Of all of the vast business of the House of Delegates conducted in Chicago in June, which included 264 items, 147 items (66%) were solely for the benefit of the public, and only 51 items (19%) solely for the benefit of doctors (*all* doctors—not just members). The remaining 66 items (25%) benefited both.

Nothing in the large agenda consumed as much of the Association's time as AIDS. For some months the Board of Trustees has been assembling data and testimony on HIV infection, which culminated in its report YY. After further discussion in reference committee and on the floor of the House, the version published elsewhere in this issue of the *Journal* was adopted. Its 17 recommendations are, said a representative of the Surgeon-General, a definitive work on which public policy can be based. The report is characterized by an overriding concern that there be a judicious balance between the well-being of those with HIV infection and protection of the public.

Though the AIDS epidemic offers almost the ultimate challenge, helping meet it is the sort of thing the AMA does best. With its superb journal it is a clearinghouse for current authoritative information. The AMA does not do everything right, of course, and in fact its massive bureaucracy can sometimes become a source of painful frustration for those of us who on occasion work within the system, but it can when it must quickly mobilize its impressive faculties and perform magnificent feats. After all, you have to take it all together, and on balance the AMA is a marvelous organization that deserves the support of every doctor. Most of its objectionable features would then doubtless vanish.

Though there are many things wrong with today's practice climate, over which we have no control, there is nothing wrong with medicine itself that collectively we cannot cure. If we doctors had practiced prevention in our own house, our practice climate might be better today. For the sake of the future, we need to be sure and take a lesson from the past.

J.B.T.

To a Waterfall

Ordinarily, going up is harder than coming down; it gives you the puffs and strains your calf muscles, among other things. But suffering from rock slides since I was there last, the already steep trail down to the bottom of Fall Creek Falls in a state park of the same name in Bledsoe and Van

Buren counties depends in places upon tree blazes to announce its presence; it keeps you on your toes, so to speak, jamming them down into your shoes and trying out your ankle tendons for durability and tensile strength. The more or less dilapidated trail down to the Cane Creek Cascades boasts the most magnificent stand of poison ivy I have ever seen, and though I tried to warn the herd of young'uns that were galloping through it that it would give them itchy bumps, most of them paid scant heed. I've wondered how they fared later on; by paying careful attention, I managed to avoid any, but it wasn't easy. That keeps you on your toes, too.

Undaunted by the threat of sore muscles, a sprain or so, and weeping skin, I took on both trails on a beautiful, warm, but not stifling day last week, and won—nary a bleb or sprain, and only a little bit of stiffness in trade for a marvelously revitalizing expedition (when one reaches my age it becomes more of an expedition than a jaunt, though I seemed to be bearing up as well on the falls trail as the younger ones. I was about to say "PTL," but since I'm not sure of its connotation anymore, I'll say "Hallelujah" instead).

What I really say "Hallelujah" about is that I live in Tennessee, where such grandeur can be an everyday experience, if you like, and even if it isn't every day, whatever it is is way ahead of the everyday experience of the biggest part of our country's couple of hundred million or so citizens. If you aren't so inclined, or have tired blood or achy joints, you don't have to hike down below the falls or to the cascades to enjoy the park, or even the falls; you can ride up to the overlook and rest in the cool shade as you watch the water plunge over the ledge to the pool a couple of hundred feet or so below. If you are looking for tranquility, the pool below the cascades is no place to go, as it is overrun by the young and nimble. But no matter. If your joints creak, there are tranquil trails that are nearly flat. And that park is just one of many such places in the state. So why would anyone want to retire to Florida or anywhere else, and leave Tennessee?

I read a note the other day in my favorite information source, the *Wall Street Journal,* that corporate acquisitions are accelerating the pace of employee relocations. When Delta Airlines acquired Western Airlines in April, for example, it moved 1,400 people within six weeks. The Employees Relocation Council says companies will transfer 6% more workers this year than last, and the moving industry has had its busiest June ever.

Despite its ever-compounding burden of bureaucratic paperwork, there are still many things to be said for the practice of medicine. It is not inconsequential that you can still, for the most part, call your shots—and you can live where you please. Tennessee is bound to please you—for lots of reasons. The next time the PRO is on your case, take a minute to think about that. Try trading the pain in wherever it is the PRO gives it to you for some pain lower down—in your calf muscles—and enjoy our state.

J.B.T.

War Is Peace

When Noah beached on Ararat
He found that he had saved a cat—
Or rather two, from where the flood
Had left the world submerged in mud.

In making up his list he'd winked;
He thought he'd make the cat extinct,
Because he knew and knew full well
Its tendency for raising hell.

But somehow in the rainy dark
Two cats had slipped aboard the ark,
And had triumphantly survived—
Malicious, sly, and many lived.

Ah, well, said Noah—that is that!
And put a curse upon the cat,
A complex triple jointed curse
That can't be reproduced in verse.

And that is why the cat will play
And frisk and frolic all the day,
And lie beside the fire and purr
And let the children stroke its fur.

But it remembers after dark
The curse it heard on Noah's ark,
And howls in ecstasy of fright
A hymn of hate throughout the night.

The Cat
Author unknown (to me)

The distaff member of a couple of my acquaintance has one of the most magnificent soprano voices I know of anywhere—on or off the operatic stage. Her husband, on the other hand, moves his voice up and down eratically and unpredictably, and since it is a powerful voice, the consequences can be devastating in group singing. Being devout Christians, they frequently engage in such activities, to the sometime consternation of those about them; it bothers them not at all, since they are persuaded that God sees past such minor arberrations into the inner person. So far as I know, their hearts are purer than most, and in tune.

But not their harmony. Near the end of his long, illustrious career, Beethoven, the paragon of instrumental composers, the master concert pianist, now stone deaf, wrote what stands in the estimation of symphonic aficionados as the paragon of symphonies, his colossal Ninth, or Choral, Symphony. Though it is magnificent in its orchestration, it is its fourth movement, with its magnificent setting of Schiller's *Ode to Joy*, that sets it apart.

Having in his own mind, not to mention those of others, conquered the world of instrumental music, Beethoven confided that he could go no further without the one instrument that surpassed all others; that instrument was the human voice. Since by that time he could hear nothing at all, the music is vocally tremendously demanding, particularly of the soprano soloist, but it is even more a tribute to Beethoven's genuis that properly sung it is music unsurpassed. One of his last public appearances was to conduct its premier; unable to hear the tumultuous applause that followed a brief moment of stunned silence at its close, Beethoven believed he had failed in his efforts, until the concertmaster turned him about to witness the prolonged standing ovation the symphony evoked.

There are those who posit that since musical instruments are a human invention, they are consequently not allowable in the worship of God. Since that is not a debatable subject with the convinced, I shall skip it, to allow, with Beethoven, that however that may be, there is no question as to who created the human voice, and that it can be a marvelous tribute to its Creator.

It can also work the other way 'round. When I say that, I am not speaking of my gravel-voiced friend. Though his singing is an aesthetic offense to my musical ear, it is uplifting to my Spirit. When I say it can also work the other way 'round, I am speaking of the hymns of hate that the voice can raise—sometimes in the name of love, and even sometimes in the name of God. Those hymns are not usually set to music, though they can be. The Ku Klux Klan maintains it only loves. Such love is the best advertisement for hate I ever saw. We need to be careful what we call love, or we may wind up, as in the great society of George Orwell's *1984*, proclaiming, "War is Peace." But I digress.

Job protested that truly we are fearfully and wonderfully made, but his understanding of anatomy and physiology was fragmentary at best. Job did not know, for example, that the voice is infinitely varied, that like the fingerprint no two are alike, and that in fact the voiceprint is the surer means of identification. The more we learn of ourselves, the more we confirm what Job discovered. On the other hand, Job knew enough to be sure of his statement. He knew enough not to substitute cacophany for harmony and squalor for beauty, and call it grand, which is so fashionable these days. (War is Peace.)

The Catechism's answer to its first question is that the chief end of man is to glorify God and enjoy him forever. I would not dare dictate what would meet the requirements toward that end, though I know some things that would not qualify, but not so many as some might think. We can praise God in ways we know not. Praise is not just standing up and singing and praying on Sunday morning. Praise involves a lot of nonvocal activities.

But praise also involves vocal activities. Though standing up there on Sunday mornings singing and praying are doubtless important parts of it, they are only a sometime activity, and the voice is used every day, for better or worse, sometimes directed toward God, but more often toward one of the least of those, his brethren. The voice can uplift, or it can crush.

What started this was not being depressed or outraged by a hymn of hate (either cat or KKK), but by listening to Strauss' *Gypsy Baron* operetta. It is uplifting. Actually, our cat emits only loving, uplifting sounds, too, though some of those that come around nights do otherwise. The more or less pejorative poem that heads this piece was obviously not written by a cat lover (his name escapes me; I memorized his poem many years ago). It describes a use of the voice that is neither praise nor praiseworthy (except maybe to other cats). The poem isn't praise, either, though it is sort of uplifting, unless maybe you are a cat.

Though man, at least theoretically, has been given more powers of discrimination than the cat, one cannot always tell it. Man has also been given by his Creator a certain amount of autonomy; he can choose to use or not to use his voice in praise, or to use it otherwise. There are a lot of otherwise uses going around; there are too many hymns of hate being sung, and not all of them by cats, either (nor cats *and* the KKK). If all voices that God made were used as intended, it might not solve the world's problems, but then again, it might. Who knows? It has never been tried. It ought to be.

J.B.T.

in memoriam

Thomas L. Ray, age 63. Died July 7, 1987. Graduate of University of Tennessee College of Medicine. Member of Knoxville Academy of Medicine.

Alvis D. Simmons, age 80. Died June 30, 1987. Graduate of University of Tennessee College of Medicine. Member of Knoxville Academy of Medicine.

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

COCKE COUNTY MEDICAL SOCIETY
H. Kenneth Johnson II, M.D., Newport

CONSOLIDATED MEDICAL ASSEMBLY OF WEST TENNESSEE
Ronald F. Taylor, M.D., Kingston Springs

KNOXVILLE ACADEMY OF MEDICINE
Joseph M. Googe, Jr., M.D., Knoxville
Joseph L. Thompson, III, M.D., Knoxville

NASHVILLE ACADEMY OF MEDICINE
Mark D. Glazer, M.D., Nashville
Michael O. Koch, M.D., Nashville
David M. Rowe, M.D., Nashville

personal news

Fred A. Guthrie, M.D., Crossville, has been certified as a diplomate of the American Board of Emergency Medicine.

Reinaldo Olaechea, M.D., Crossville, has been initiated as a fellow of the International College of Surgeons.

Thomas W. Orcutt, M.D., Nashville, has been named as councilor for the American College of Surgeons for the Middle Tennessee region.

The following TMA members have been initiated as fellows of the American Academy of Facial Plastic and Reconstructive Surgery: *Stanley L. Bise, M.D.,* Crossville; and *Charles W. Cox, M.D.,* Jackson.

TMA Members Receive AMA Physician's Recognition Award

Twenty-eight TMA members qualified for the AMA Physician's Recognition Award during June 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

John W. Avera, M.D., Oak Ridge
David W. Bell, M.D. Memphis
Samuel S. Binder, M.D., Chattanooga
Dee J. Canale, M.D., Memphis
David C. Chaffin, M.D., Cleveland
Cully A. Cobb, Jr., M.D., Nashville
Thomas W. Conway, M.D., Newport
Bennett Y. Cowan, Jr., M.D., Bristol
Tommy H. Crunk, M.D., Springfield
Doran D. Edwards, M.D., Madison
Sandra A. Eisele, M.D., Knoxville
Thomas C. Farrar, M.D., Nashville
Howard R. Foreman, M.D., Nashville
Alan S. Henson, M.D., Madison
Edwin C. Jordan, M.D., Clarksville
Albert L. Maduska, M.D., Memphis
Daniel C. Martin, M.D., Memphis
William R. McKissick, M.D., Knoxville
John S. McNulty, M.D., Cleveland
Robert W. Meadows, M.D., Knoxville
Charles S. Mitchell, M.D., Oak Ridge
Alan M. Nadel, M.D., Memphis
James W. Ness, M.D., Tellico Plains
C. Leon Partain, M.D., Nashville
Patricia F. Robinson, M.D., Nashville
Deborah J. S. Ruark, M.D., Hermitage
Marion B. Tallent, Jr., M.D., Nashville
Frank T. Varney, M.D., Johnson City

announcements

CALENDAR OF MEETINGS

NATIONAL

Oct. 10-13	International Symposium on Calcium Antagonists: Pharmacology and Clinical Research (sponsored by NY Acad Sci)—New York
Oct. 10-14	American Society of Anesthesiologists—Atlanta
Oct. 11-16	American College of Surgeons—San Francisco Hilton
Oct. 12-14	Society for Medical Decision Making—

	Sheraton Society Hill Hotel, Philadelphia
Oct. 15-18	American Association of Electromyography and Electrodiagnosis—Hyatt Regency, San Antonio, Tex.
Oct. 15-18	American Society of Internal Medicine—J. W. Marriott, Washington, D.C.
Oct. 18-21	American Neurological Association—Hilton & Towers, San Francisco
Oct. 18-22	American Public Health Association—New Orleans
Oct. 18-22	International Health Society—New Orleans
Oct. 18-23	American Academy of Physical Medicine and Rehabilitation—Marriott World Center, Orlando, Fla.
Oct. 19-23	American Academy of Occupational Medicine—Hyatt Regency, San Francisco
Oct. 20-25	American Academy of Neurological and Orthopaedic Surgeons—Alexis Park Resort Hotel, Las Vegas
Oct. 21-25	American Academy of Child Psychiatry—Sheraton, Washington, D.C.
Oct. 22-24	Child Neurology Society—Vacation Village, San Diego
Oct. 23-29	American Society for Therapeutic Radiology and Oncology—Sheraton, Boston
Oct. 25-28	American Association for the Study of Liver Disease—Marriott, Chicago
Oct. 25-28	Medical Group Management Association—Sheraton, Washington, D.C.
Oct. 25-30	Congress on Neurological Surgeons—Convention Center, Baltimore
Oct. 26-28	American College of Gastroenterology—Fairmont Hotel, Dallas
Oct. 26-30	American College of Chest Physicians—Marriott, Atlanta
Oct. 27-Nov. 1	American Medical Women's Association—Buena Vista Palace, Orlando, Fla.
Nov. 1-4	American Physicians Art Association Meeting and Exhibit—San Antonio, Tex.
Nov. 1-4	Southern Medical Association—San Antonio, Tex.
Nov. 2-5	Interstate Postgraduate Medical Association—The Diplomat, Hollywood, Fla.
Nov. 2-7	American College of Emergency Physicians—San Francisco
Nov. 4-7	American Cancer Society—New York
Nov. 4-7	American Medical Writers Association—Drake Hotel, Chicago
Nov. 4-8	American Society of Cytology—New Orleans
Nov. 5-7	Society for the Scientific Study of Sex—Westin Peachtree Plaza, Atlanta
Nov. 6-7	American Association of Neurological Surgeons and Congress of Neurological Surgeons Joint Section on Trauma—Marriott Orlando World Center, Orlando, Fla.
Nov. 6-11	National Perinatal Association—Adam's Mark Hotel, St. Louis
Nov. 7-12	American Association of Blood Banks—Orlando, Fla.
Nov. 7-12	Association of American Medical Colleges—Washington Hilton, Washington, D.C.
Nov. 8-10	Association of American Physicians and Surgeons—New Orleans
Nov. 8-10	Association for Hospital Medical Education—Washington, D.C.
Nov. 8-12	American Academy of Ophthalmology—Dallas
Nov. 8-13	American Society of Maxillofacial Surgeons—Georgia World Congress Center, Atlanta
Nov. 8-13	American Society of Plastic and Reconstructive Surgeons—Atlanta
Nov. 8-13	Association of Military Surgeons of the United States—Las Vegas
Nov. 10-13	Association for Medical Education and Research in Substance Abuse—Crowne Plaza Hotel, Rockville, Md.
Nov. 11-15	Academy of Psychosomatic Medicine—Riviera Hotel, Las Vegas
Nov. 11-15	Christian Medical Foundation International—Tampa, Fla.
Nov. 11-15	International Congress of Gynecologic Endoscopy—Sheraton Palace, San Francisco
Nov. 12-14	Association of Clinical Scientists—Tampa, Fla.
Nov. 14	Association for the Advancement of Psychotherapy—New York
Nov. 14-18	American College of Allergists—Marriott Hotel/Copley Plaza, Boston
Nov. 16-18	American Society of Bariatric Physicians—Bally's Hotel, Las Vegas
Nov. 16-19	American Heart Association—Convention Center, Anaheim, Calif.
Nov. 18-22	Gerontological Society of America—Washington Hilton, Washington, D.C.
Nov. 21	Chinese-American Medical Society—New York
Nov. 28-Dec. 4	Radiological Society of North America—Palmer House, Chicago

STATE

Oct. 9-10	Tennessee Pediatric Society—Chattanooga Choo-Choo
Nov. 3-6	Tennessee Academy of Family Physicians 39th Annual Scientific Assembly—Gatlinburg
Nov. 13-15	Tennessee Psychiatric Association Fall Meeting (in conjunction with American Society for Adolescent Psychiatry)—Vanderbilt Plaza Hotel, Nashville.

Russell E. Miller, Jr. Named TMA Executive Assistant

Tennessee Medical Association Executive Director L. Hadley Williams has announced the appointment of Russell E. Miller, Jr. as Executive Assistant. Miller, 23, is a native Nashvillian and holds a bachelor of arts degree in public relations/speech communications from Auburn University accompanied by minors in journalism and industrial product design.

Before graduation from Auburn in 1986, Miller was a sports correspondent to the Associated Press Wire Service and news writer for a local Auburn radio station.

Mr. Miller joins TMA having previously been an advertising account executive with an area magazine and assistant buyer for a local retailer.

"High Quadriplegia– The Ultimate Challenge"

Plan to attend:

A two-day symposium at Colony Square Atlanta

April 7-8, 1988

Registration: $225

Symposium
Co-Chairmen

David F. Apple, Jr., M.D.
Medical Director

Donald P. Leslie, M.D.
Medical Director
High Quadriplegia Program

A medical symposium addressing the acute and rehabilitative care of the C-1 through C-4 high quadriplegic. Hosted by Shepherd Spinal Center in Atlanta, now the nation's largest dedicated spinal cord injury hospital. Issues to be investigated include: medical, psychosocial and high tech approaches to care and rehabilitation. Special emphasis on ventilator weaning, the interdisciplinary care approach, phrenic nerve pacer implants and community reintegration.

Symposium Preview:

High Quadriplegics: They Can Go Home Again

With high quadriplegics surviving at unprecedented rates, quality of life issues and discharge planning are of paramount importance from the first day of admission to the specialty setting. The philosophy of treatment at SSC will be covered, including the referring physician's role in long-term medical management.

Medical Overview: Care of the High Quadriplegic

The potential for complications such as deep vein thrombosis, stress ulceration, decubitus, pneumonia, urinary tract infections and sepsis poses a serious threat to high quadriplegic patients. Prevention strategies, the benefits of early mobilization of ventilator dependent patients and medical management of complications are covered.

Ventilator Weaning

All high quadriplegics at Shepherd Spinal Center are evaluated to determine their candidacy for phrenic nerve pacer implants and their potential for weaning from mechanical ventilation. The pulmonary evaluation studies performed at SSC and protocols for weaning are included.

Panel and Concurrent Session Topics:

- Pulmonary Issues
- Social Work: Discharge Planning, Peer Support, Sexuality
- The Therapeutic Value of Sensory Experience
- The Biofeedback Program at SSC
- Ventilator Home Care
- Focus On: Phrenic Pacer Implantation
- Departmental Presentations by O.T., P.T., Recreation Therapy, Social Work, Respiratory Care, Education, Nutritionists
- Emphasis on specialized equipment

For Physicians Only:

Grand Rounds at Shepherd Spinal Center

REGISTRATION IS LIMITED. Reserve your space today, by sending a check for $225, payable to Shepherd Spinal Center, to: Lesley M. Hudson, Symposium Registrar, Shepherd Spinal Center, 2020 Peachtree Road, N.W., Atlanta, GA 30309. Confirmations of early registrations and a symposium information packet will be mailed in October.

High Quadriplegia– The Ultimate Challenge

Name______________________________

Specialty____________________________

Address_____________________________

City__________________State________Zip________

Check one: ☐ Check enclosed. Reserve my space now. ☐ Please send a complete information packet.

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Primary Malignant Melanoma of Pineal Region

BRIAN R. CARLSON, M.D.; ALAN D. GLICK, M.D.; and
ARTHUR R. CUSHMAN, M.D.

Malignant melanoma is seldom primary in the central nervous system (CNS), and involvement of the pineal region is rare.[1] We describe the unusual findings of primary CNS malignant melanoma of the pineal region with electron microscopy studies.

Report of a Case

This 77-year-old white woman was admitted on Sept. 18, 1984 with a one-week history of poor memory and inability to walk, with episodes of falling. There was no history of abnormal pigmented skin or eye lesions.

On physical examination the patient was awake, oriented, and cooperative. The pupils showed evidence of previous cataract surgery; no papilledema was identified. Her cranial nerves were intact, and though muscular strength appeared normal bilaterally, she was clumsy and could not walk. According to her family she was confused. No abnormal pigmented skin lesions or abnormal pigmented lesions of the eye were identified.

A CT scan of the head revealed a mass in the pineal gland region thought to be producing obstructive hydrocephalus with dilatation of the lateral ventricles and third ventricle. Carotid and vertebral arteriograms showed a faint blush in the pineal gland region. The lateral ventricles were thought to be dilated.

During the two days after a ventricular peritoneal shunt was performed on Oct. 2, 1984, a subdural hematoma developed on the right, for which a right craniotomy was performed two days later; the leptomeninges were found black and slightly thickened. The hematoma was evacuated and a biopsy of the pigmented leptomeninges was performed, showing leptomeningeal melanosis, which in conjunction with the pineal region mass was thought to indicate clinically an intracerebral malignant melanoma arising in the pineal region.

The patient recovered from the operation but then developed multiple medical problems including bronchopneumonia and subclavicular abscess, and died on Oct. 11, 1984 with sepsis.

All of the relatively few skin lesions found at postmortem examination were examined microscopically. They showed changes of lentigo simplex, seborrheic keratosis, epidermal cyst, intradermal nevus, acrochordon, and lymphangioma. No primary or metastatic malignant tumor was identified in any of the skin lesions.

No evidence of malignant melanoma was identified outside of the CNS. The left orbital contents of the left eye were examined and no malignant tumor or pigmented lesions were identified.

The fresh brain weighed 1,380 gm. Although minimally thickened, all the leptomeninges were black, including those of the cerebrum, cerebellum, brainstem, and spinal cord. The ventricles also contained focal black areas measuring up to 2 mm, but no definite masses were identified in the leptomeninges or ventricles. The pineal body was completely replaced by a black, gray, and tan tumor measuring 3.0 × 2.5 × 1.5 cm, which completely occluded the cerebral aqueduct and invaded into the inferior colliculus (Fig. 1). The third ventricle and lateral ventricles were dilated with changes of hydrocephalus.

Microscopically the cellular tumor replacing the pineal gland was composed of plump spindle cells with variable amounts of eosinophilic cytoplasm tending to form interlacing fascicles (Fig. 2); individual cellular outlines were indistinct. The nuclei tended to be elongate, and some were vesicular with prominent nucleoli, whereas others were hyperchromatic. Both fine granules and coarse clumps of melanin pigment could be identified in many of the tumor cells, and fontana stains for melanin were positive. The pigmented zones of the ventricles contained similar melanin-containing neoplastic cells. The leptomeninges of the cerebral cortex, brainstem, and spinal cord were diffusely involved by melanin-laden cells, many of which were cytologically bland, but neoplastic cells like those in the pineal region mass could be identified. Though the tumor cells tended to invade the

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From the Departments of Pathology (Dr. Carlson) and Neurosurgery (Dr. Cushman), HCA Donelson Hospital, and the Department of Pathology (Dr. Glick), Vanderbilt University Medical Center, Nashville.

Reprint requests to Department of Pathology, HCA Donelson Hospital, 3055 Lebanon Road, Donelson, TN 37214 (Dr. Carlson).

underlying CNS tissue along blood vessels, the only tumor mass was in the pineal region. Electron microscopy showed the tumor cells of the pineal tumor to contain numerous melanosomes in all stages of development (Fig. 3).

Comment

This patient had a primary malignant melanoma of the pineal region of the CNS; the only definite mass identified at postmortem examination had replaced the pineal body. We excluded to the best of our ability primary malignant melanoma outside the CNS clinically and by postmortem examination. No other masses were found in the CNS, although ventricular involvement was identified and the leptomeninges were extensively pigmented. Neither numerous skin nevi, very large nevi, nor ocular melanocytic nevi were present.

Six previous cases of primary malignant melanoma of the CNS and pineal region have been recorded.[1-3] Usually involvement of the pineal region has been associated with additional CNS tumor nodules of varying sizes,[2,3] though Arlant et al[1] described a primary malignant melanoma of the pineal region very similar to our own case.

It is likely that malignant melanoma of the pineal region arises from melanin-containing cells normally found in the leptomeninges, particularly in the region of the spinal cord and ventral aspect of the brain.[2-5] Gibson[2] gives reference to melanoblasts seen in the pia mater of the interlobular septa that cross the pineal gland in some animals. The pigmented changes in this patient's leptomeninges were widespread with the leptomeninges of the CNS thickened by cytological bland sheets of intensely pigmented cells. The changes were those of leptomeningeal melanosis. Whether the leptomeningeal changes represented generalized melanocytic changes of the leptomeninges that preceded the pineal region tumor or leptomeningeal spread via the cerebrospinal fluid of preceding pineal primary malignant melanoma is indeterminate. It may be that the malignant melanoma arose from the diffusely pigmented leptomeninges in the region of the pineal gland, with subsequent invasion and destruction of the pineal gland, since such behavior is characteristic of malignant melanoma arising elsewhere, and correlates with the relatively bland appearance of the pigmented leptomeninges. The noninvasive melanocytic lesions may have played a role analogous to nevi of the skin. On the other hand, it is possible that the tumor arose in the pineal region with subsequent diffuse metastatic spread through the ventricles and leptomeninges. Primary pineal region tumors are well known to disseminate widely in the leptomeninges via the

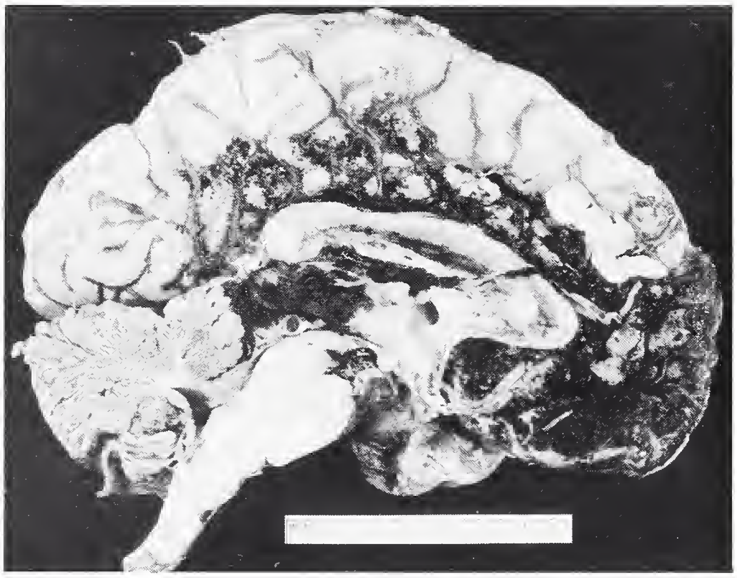

Figure 1. Sagittal sections showing mottled black and gray tumor in the pineal region. Pigment deposits are identified in the leptomeninges and ventricles.

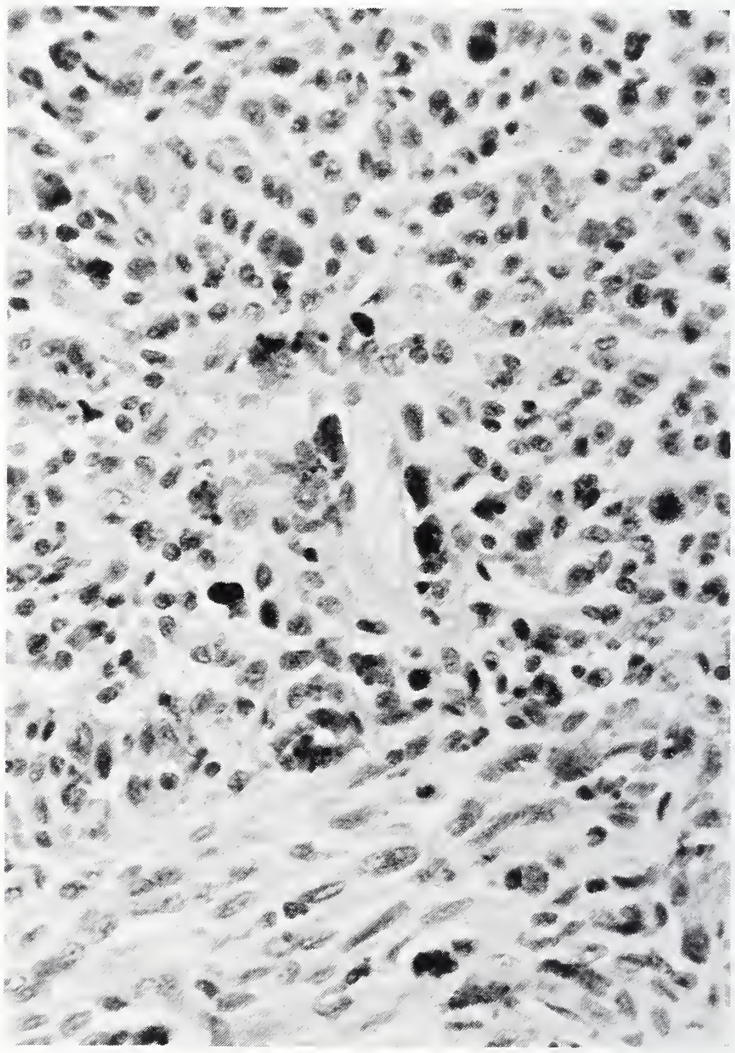

Figure 2. Tumor cells, some of which contain melanin pigment (hematoxylin-eosin, X400).

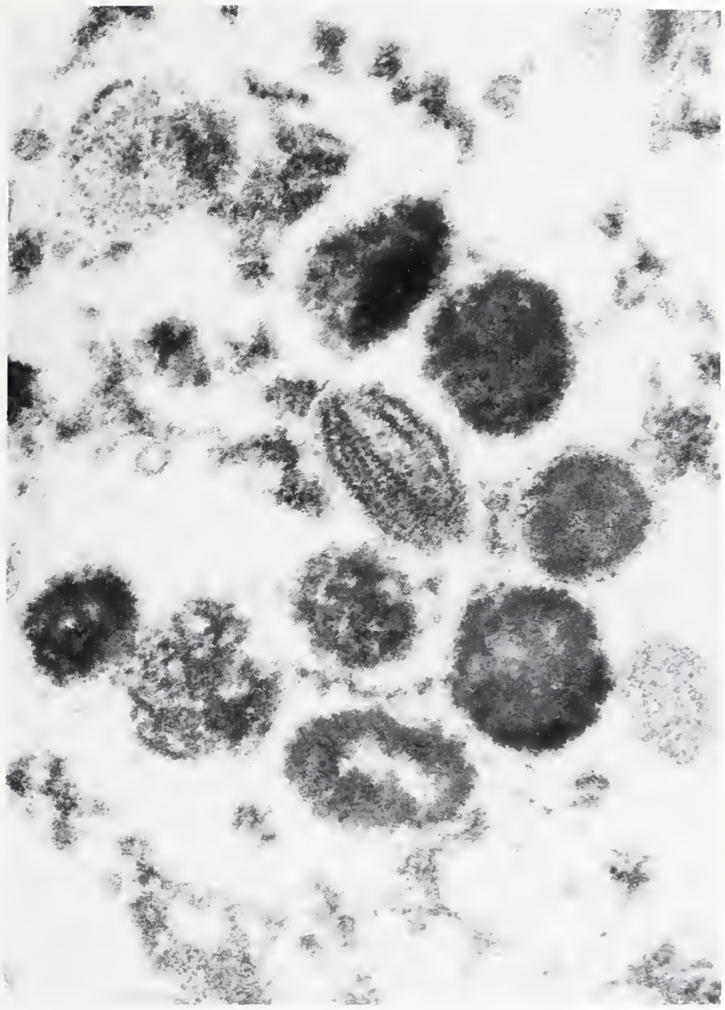

Figure 3. Cytoplasm of tumor cell, containing stage III and IV melanosomes (lead citrate, uranyl acetate, X91,000).

cerebrospinal fluid.[6-8] The ventricular lesions are almost certainly metastatic, and would suggest such an occurrence. Based on our findings, either or both processes are possible.

Patients with malignant melanoma of the pineal region have done poorly, with death occurring within 12 days to four months after admission to the hospital.[1-3]

The anatomic location of the pineal gland essentially precludes total excision of any neoplasm in this area, and a condition such as malignant melanoma would be expected to cause death rapidly.

In summary, we have briefly described our experience with this extremely rare tumor, including electron microscopy studies. Malignant melanoma should be considered in the differential diagnosis of a mass in the pineal region, especially when associated with pigmented leptomeninges.

Acknowledgments:

We wish to thank W. Geoffrey Hartmann for assistance with the photomicrographs and Phyllis Elftmann for her secretarial assistance.

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Autotransplantation for Multiple Renal Lesions

THOMAS G. PETERS, M.D.

Renal autotransplantation has been successfully applied to treat a number of disease processes for over two decades. By 1975, ex-vivo surgery had been successfully used for kidney trauma, renal carcinoma, renal arterial lesions, ureteral surgery, and renal stone disease.[1] Ex-vivo preservation and autotransplantation became recognized as a preferred method to conserve renal function in conjunction with repair of abdominal aortic aneurysms associated with renal arterial disease, in treatment of renal pelvis disorders, and in pediatric renovascular disease.[1-3]

Within the last decade, autotransplantation has been more widely applied to microvascular repair of renal arterial lesions, and has been effective in large series of patients with hypertension from both simple and complex renal arterial lesions.[4-6] In cases of compromised renal function, or single but diseased kidney, ex-vivo preservation and autotransplantation is emerging as the method of choice when renal corrective surgery is necessary.[7,8] Successful ex-vivo surgery for multiple renal lesions was undertaken at the Memphis Veterans Administration Medical Center to treat a patient with long-standing hypertension, kidney stone disease, and renal insufficiency.

Case Report

A 66-year-old white man was first diagnosed with hypertension in 1968. Medical therapy was initiated then, and in 1985 angioplasty of the left renal artery was performed for uncontrollable renovascular hypertension related to atherosclerotic disease. The successful angioplasty made blood pressure control easier, but treatment was still necessary, requiring five oral medications. Dyspnea and orthopnea accompanied his worsening hypertension, and ultimately his blood pressure could be controlled only with medications and bed rest.

He was admitted to the Memphis VAMC for reevaluation of renovascular hypertension in February of 1987, where additional history disclosed 30-pack years of tobacco abuse, mild chronic obstructive bronchopulmonary disease, and a prior coronary artery bypass procedure. A pulmonary embolus in 1985 ultimately required placement of a vena cava umbrella. The patient had previously tolerated replacement of the left hip with a prosthetic device, hemorrhoidectomy, and a right inguinal hernia repair. Bacteriuria or mild pyuria had been present on several occasions in the past, and he had been treated for recurrent urinary tract infections. A brother had died of hypertension at 49 years of age.

The blood pressure on admission was 178/110 mm Hg. The fundi disclosed arteriovenous nicking, and there were no neck bruits. Bilateral pulmonary rales were present posteriorly and an S-3 gallop was heard. The abdomen and anterior chest had well-healed surgical scars. Moderate pretibial edema and slight arthritic wrist deformities were present bilaterally. The remainder of the physical examination was negative.

The blood pressure ranged from 160/76 to 240/148 mm Hg during early hospitalization. The highest serum creatinine of 3.7 mg/dl and blood urea nitrogen (BUN) of 77 mg/dl improved with bed rest and intensive treatment for hypertension (serum creatinine 2.3 mg/dl; BUN 40 mg/dl), but never normalized preoperatively. Renal angiogram showed a tight stenotic lesion at the takeoff of the right renal artery (Fig. 1), and a renal scan showed severely diminished renal function bilaterally (Fig. 2). Renal vein renins were 24.9 ng/ml/hr on the left and 50.5 ng/ml/hr on the right; lower inferior vena caval renin (systemic) sample was 16.5 ng/ml/hr. Examination of additional angiogram films also disclosed a renal stone in the lower portion of the right renal pelvis. The conclusion of the investigation was that a renal stone, severe renal artery stenosis, and possibly other lesions were present. Since renal insufficiency was continuous, the proposition that normal left kidney function followed the prior angioplasty was suspect; therefore, operation was planned as though the right kidney were the only potentially normal renal mass.

On Feb. 27, 1987, through a long right curvilinear lower abdominal incision the right kidney, ureter, and renal vein were fully isolated and the vena cava was mobilized to obtain optimal length of the renal artery. The vena cava was patent above and below the previously placed umbrella. A 5-mm aneurysm was found at the super pole of the right renal artery at its bifurcation.

The external iliac artery and vein were next isolated from surrounding structures to assure patency of both vessels, and the ureter was clamped below the pelvic inlet and divided. With clamps on the renal artery and vein, they were divided and the kidney was removed and flushed with heparinized Ringer's lactate solution at 4°C until the effluent was clear and core cooling was obtained. Under protection of adequate cold preservation, a pelvolithotomy allowed removal of a 2 × 1-cm kidney stone; a watertight closure followed.

Next, after the renal artery aneurysm was resected with a cuff adequate to secure the arterial anastomosis, orthotopic renal autotransplant was performed (Fig. 3) leaving a small closed-system drain posterior to the kidney near the pelvolithotomy. Approximately one hour after removal of the kidney, the blood pressure dropped precipitously but responded to intravenous fluids within minutes. Careful fluid balance without antihypertensive agents controlled the patient's blood

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From the Surgical Service, Veterans Administration Medical Center, Memphis.

Reprint requests to Surgical Service, VAMC, 1030 Jefferson Ave., Memphis, TN 38104 (Dr. Peters).

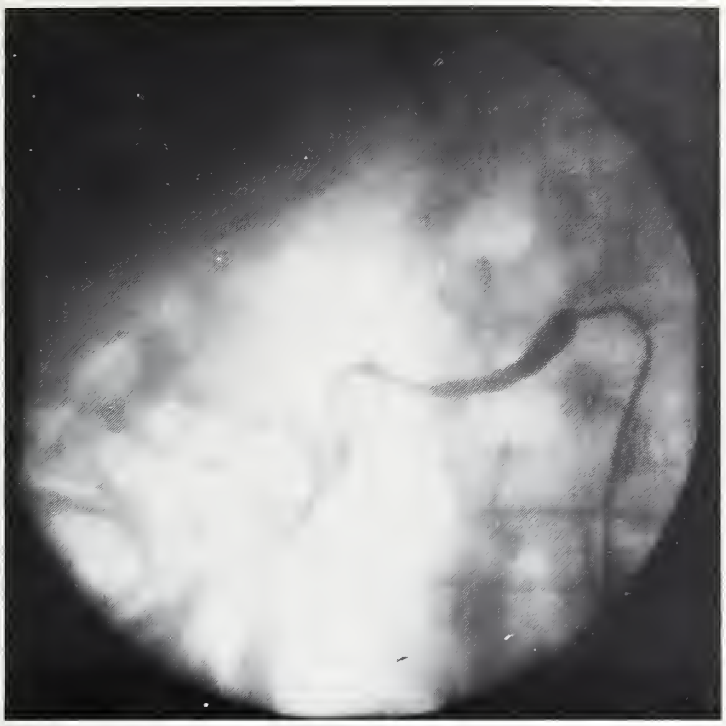

Figure 1. Selective right renal arteriogram showing severe stenosis (at catheter tip), poststenotic dilation, and a small aneurysm (unconfirmed until operation) at the superior aspect of the renal artery bifurcation.

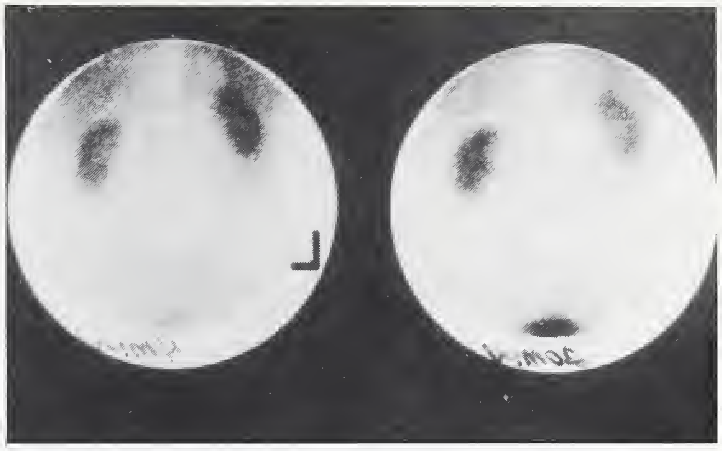

Figure 2. Renal scan (Tc-DTPA) shows no excretion at 4 minutes, and principally right renal functional excretion at 20 minutes (labels anatomically correct, original scans labeled on reverse side).

pressure during the remainder of surgery and in the immediate postoperative period.

Wound healing occurred without incident, and on the fourth postoperative day a renal scan disclosed prompt return of function of the autotransplanted kidney (Fig. 4); serum creatinine decreased to 2.0 mg/dl and BUN to 35 mg/dl at the time of discharge on the tenth postoperative day. Following discharge, blood pressure of 120/60 mm Hg was maintained by a single diuretic tablet daily and low doses of methyldopa. Renal function slowly improved as serum creatinine and BUN values became normal within the first six weeks postoperatively.

Discussion

Autotransplantation has been successful for a variety of renal lesions, especially when there is renal insufficiency or the contralateral kidney is absent. Diminished renal function has been a compelling reason to use ex-vivo preservation when trauma, malignancy, vascular lesions, stone disease, and other problems could be successfully approached only by careful removal of the kidney, preservation of its function by cold perfusion, and correction of the abnormality by exvivo surgery.[1,2,4-8] These techniques are now more widely applied with multiple renal lesions, as well as in conjunction with treatment of other significant vascular lesions such as abdominal aortic aneurysms involving one or more renal arteries.

Two large series have compared the improved results of renal autotransplantation with other surgical approaches to renovascular hypertension. In 1982, Belzer et al[5] reported 34 cases of ex-vivo renal surgery performed for occlusive renal vascular lesions, aneurysmal vascular disease, or both. The 86% cure of renovascular hypertension unaccompanied by mortality or renal functional loss related directly to ex-vivo preservation or renal autotransplantation. In 1985, Dubernard et al[6] reported on 78 patients treated for renovascular hypertension by operative techniques. There was a 96% cure without technical failure in 33 patients in whom autotransplantation was used. The 80% cure in 45 patients treated by bypass surgery in that series was not only less successful, but was accompanied by technical complications related to vascular interposition graft failure and protracted hypertension. These

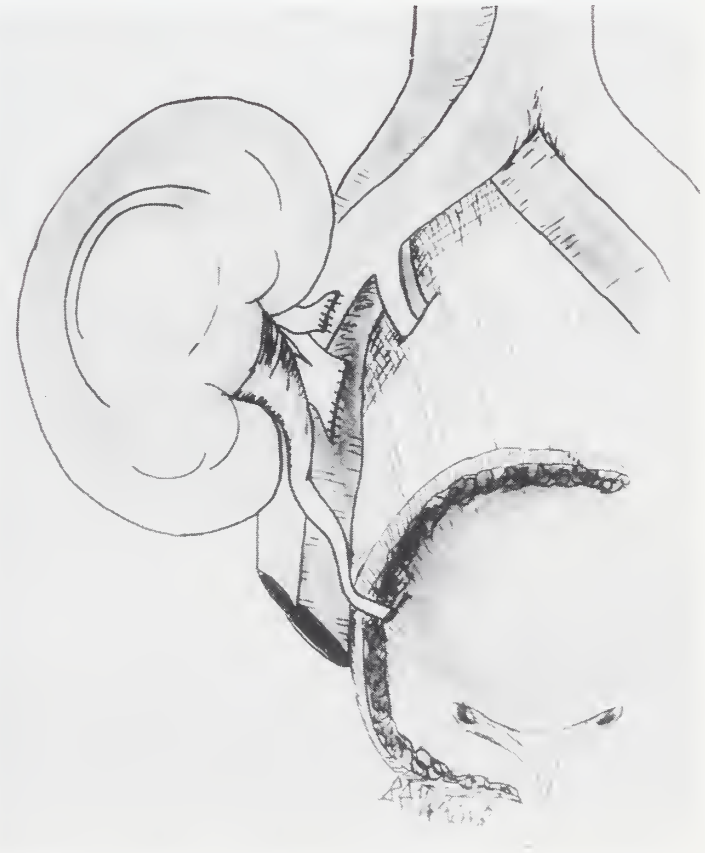

Figure 3. Orthotopic renal autotransplant with end-to-side renal to external iliac arterial and venous anastomoses, and a tunneled ureteroneocystostomy.

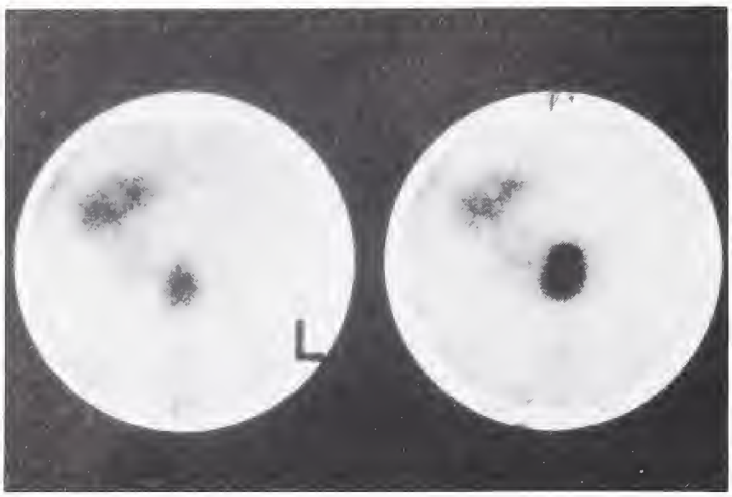

Figure 4. Autotransplant renal scan (Tc-DTPA) showing normal function at 10 and 30 minutes.

two reports make clear the proposition that fear of technical failure accompanying autotransplantation is unwarranted, and that successful therapy for simple renovascular lesions may be most often accomplished using ex-vivo preservation and autotransplantation.

It now is accepted that some neoplasms of the kidney or ureter as well as certain cases of renal stone disease can best be treated by ex-vivo renal surgery and autotransplantation.[1,2,7,8] In addition, vascular or parenchymal trauma of the kidney may sometimes best be repaired with the kidney cooled and taken to the back table; microvascular surgical techniques can be used in such circumstances as well as in the repair of intrarenal vascular disease, arteriovenous fistula, and reoperative renovascular lesions.[4,5,7,8] Clearly, atherosclerotic vascular disease and renal artery aneurysms can be safely approached by ex-vivo renal surgery. Complex or multiple renal lesions should probably be treated by ex-vivo techniques, since warm ischemic injury can be avoided, allowing the surgeon ample time to complete any complicated operative task. Most importantly, the solitary kidney with any of the above lesions is best treated by ex-vivo preservation autotransplantation to not only correct the lesions, but also optimize postoperative renal function.

Renal autotransplantation was successfully applied when multiple lesions of a kidney were present. This kidney represented the major portion of the patient's functional renal mass. Renal autotransplantation after ex-vivo surgery can be safely applied, and should now be considered as part of the routine surgical armamentarium in the operative treatment of multiple lesions of the same kidney or wherever renal insufficiency accompanies the need for renal corrective surgery.

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APRIL 1988

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
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10	11	12	13 TMA 153RD ANNUAL MEETING Peabody Hotel—Memphis	14	15	16
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Dissecting Aneurysm of the Coronary Artery

RICHARD C. HARRUFF, M.D., Ph.D.; C. LEE FERRELL, B.A.;
JOHN HAGAN, B.A.; H. WENTZELL HAMNER, B.A.; and DAVID HANCOCK, B.A.

Dissecting aneurysm of the coronary arteries is a rare but recognized cause of sudden death. To date, approximately 70 such cases have been reported. Smith[1] reviewed a total of 43 in 1975, and Robinowitz et al[2] in 1982 described some of the characteristic features of the disease. In this report, we present a case of sudden death caused by a dissecting aneurysm of the left anterior descending coronary artery, describing the clinical and pathologic features of this case, and compare it with those cases previously reported in order to further the recognition and understanding of the disease process.

Case Report

A 36-year-old black woman complained of sudden onset of chest pain; after a few minutes of delay while waiting for her boyfriend to arrive, she was taken by car towards the local hospital. En route, she began vomiting and became unresponsive, whereupon the boyfriend stopped and called an ambulance. Resuscitative efforts by the ambulance paramedics and by emergency room personnel failed to revive her.

Past medical history obtained from her mother indicated that the subject visited a number of physicians in the past but never had been diagnosed with any particular disease. She had, however, suffered from general fatigue and had menstrual problems. Six months prior to her death, a dilatation and curettage, performed because of dysfunctional uterine bleeding, showed "nonspecific chronic endometritis." Her last pregnancy was seven years earlier, and she was not taking oral contraceptives or any other drug.

The body was referred because no physician was willing to sign the death certificate. Autopsy, performed 20 hours after death by the medical examiner because of the suddenness and unexpectedness of the death, showed a normally developed and nourished youthful-appearing black woman whose heart weighed 310 gm and had normal configuration, chambers, valves, and myocardial appearance and texture. There was no appreciable coronary atherosclerosis and only minimal abdominal aortic atherosclerosis. In the left anterior descending coronary artery was a dissecting aneurysm that involved essentially its entire length starting at its origin from the left circumflex artery. Fresh clotted blood formed a hematoma around the media of the vessel, collapsing and compressing the lumen upon itself (Fig. 1). The hematoma did not dissect into smaller branches of the artery.

Microscopic evaluation of the organs showed mild fatty liver and mild arteriolonephrosclerosis; other organs were normal. No vasculitis or other blood vessel disease was apparent in any of the organs. The myocardium showed no recognizable ischemic changes, consistent with the suddenness of the death. Several sections of the involved left coronary artery, along with sections of the other major coronary arteries and of the thoracic and abdominal aorta were studied in detail, using the following stains in addition to hematoxylin and eosin: Masson's trichrome, Verhoeff's-van Gieson for elastic tissue, periodic acid Schiff (PAS), alcian blue, and mucicarmine. Sections of normal aortas from two women and one man, all of similar age to the subject, were stained identically for comparison.

In subserial sections of the involved left anterior descending coronary artery, the dissecting aneurysm appeared as fresh blood with few clumps of platelets and fibrin filling a defect between the media and the adventitia, associated with inward dislocation of the media and resulting in luminal collapse. There was no sign of organization. No intimal tear was seen, although there was focal hemorrhage in the media. An elastic stain showed the hematoma occupying a cleft between the

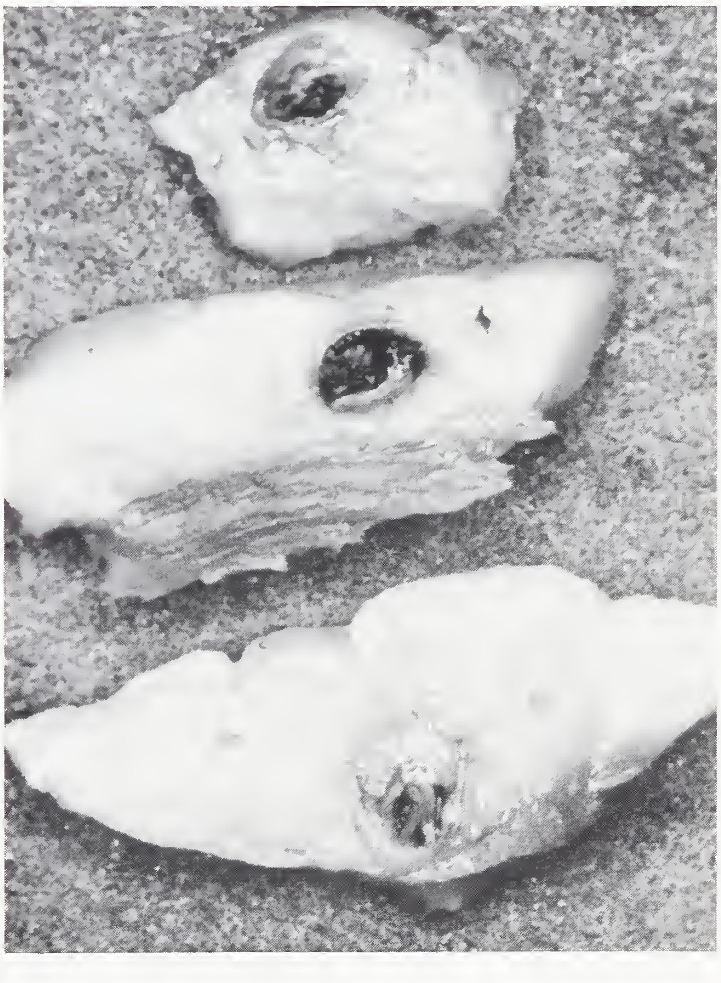

Figure 1. Representative cross sections of segments of the left anterior descending coronary artery showing hemorrhage into the wall of the vessel, hematoma formation between the media and adventitia, and resulting collapse occluding the lumen.

From the Office of the Medical Examiner, Shelby County, and the Department of Pathology, University of Tennessee, Memphis.

Reprint requests to Department of Pathology, University of Tennessee, 3 N. Dunlap St., Memphis, TN 38163 (Dr Harruff).

outmost layers of the media and the external elastic membrane; the same stain showed reduplication of the internal elastic membrane and mild intimal thickening, both in the involved left anterior descending coronary artery as well as in the other main coronary arteries.

With hematoxylin and eosin staining, the spaces between the elastic fibers in the aorta sections showed distinct vacuolization, which was accentuated by the PAS and trichrome stains. There was no vacuolization of the media in any of the coronary arteries. With both mucicarmine and alcian blue stains, there was increased uptake of stain in the subject's aorta, as compared to the normal ones. This increased stain affinity appeared to be associated with the vacuolization between the elastic fibers and was distributed throughout the width of the vessel. In the normal controls, slight mucin staining was limited to the inner third of the vessel and was not associated with vacuolization.

The adventitial adipose tissue surrounding the involved left anterior descending coronary artery was infiltrated with abundant eosinophilic granulocytes and a few mononuclear cells. No cellular infiltrates were seen in or around any of the other blood vessels.

Discussion

Primary dissecting aneurysm of the coronary arteries is a rare but increasingly recognized cause of sudden death. About 70 cases have been reported, and there have been two literature reviews.[1,2] The disease most commonly affects moderately young women, frequently in the postpartum period, and the artery involved is usually the left anterior descending coronary artery alone.[1,2] Other cases have been reported to occur in childhood,[3] and in old age on occasion to involve multiple coronary arteries,[4] in intravenous drug abusers,[5] following chest trauma,[1,2] after coronary bypass surgery and coronary angiography,[1] in Marfan syndrome,[1,2] in sarcoidosis,[6] and with fibromuscular dysplasia of the coronary arteries.[7] Although the onset of death is nearly always sudden, there is occasionally morphologic evidence of infarct, and rarely the diagnosis is made before death.[8]

Despite many of these cases having been studied microscopically in considerable detail, as we have done, the etiology of the disease remains unknown. Two morphologic features that have been frequently documented in reported cases, both of which were seen in our case, are adventitial inflammation composed primarily of eosinophilic granulocytes and increased mucoid material in the media of the affected coronary artery and/or the unaffected aorta.

The accumulation of mucoid material in the media has been likened to the cystic medionecrosis seen in dissecting aortic aneurysms.[2] It is possible that this change represents a loosening of the ground substance in the vessel wall, predisposing to dissection initiated by local hemodynamic forces. We were able to document an increase in mucoid material in the media of the aorta only, and not in the coronary arteries. It is possible, of course, that the dissection itself obliterated a focus of mucoid degeneration. The coronary arteries did show nonspecific abnormalities in that both the affected and unaffected arteries displayed reduplication of the internal elastic membranes.

Like the majority of cases in which specific mention is made regarding vascular inflammation, our case showed infiltration by large numbers of eosinophilic granulocytes limited to the adventitia associated with the dissection. The significance of these eosinophilic infiltrates is subject to debate. Robinowitz et al[2] initially proposed that the eosinophils release lytic substances from their granules, which damage the vessel wall. The alternative viewpoint, advanced by Dowling and Buja,[9] is that the eosinophilic infiltrates are reactive, attracted to the region by histamine released from the vicinity of the dissection. From our observations, we agree that it seems unlikely that eosinophilic infiltration of the adventitia alone could result in damage to the media, and we propose that mucoid material released from accumulations in an abnormal artery may be an additional factor in eliciting eosinophilic infiltration.

Because of the similarities in this case with many of those reported before, it is likely that spontaneous dissecting aneurysm of the coronary artery represents a distinct disease process, the etiology of which is unknown, but which involves an abnormality in the ground substance of the arterial media that combined with local hemodynamic forces produces the lesion.

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Medical Grand Rounds

The Uncontrolled Arrhythmia

DAVID M. MIRVIS, M.D.

Case Presentation

A 42-year-old woman was referred to the University of Tennessee Medical Center for evaluation of ventricular premature beats. One year earlier, she began to have frequent palpitations, described as single events without runs of rapid or irregular cardiac activity. Although she had neither lightheadedness nor syncope, she considered the palpitations disabling; exercise tolerance was good, without dyspnea. Previous attempts at therapy included prescription of several drugs; quinidine caused severe gastrointestinal distress with diarrhea, procainamide produced arthralgias and a positive FANA, and disopyramide induced an acute psychotic episode. There was no past history of cardiac disease, chest pain, dyspnea, or edema. Family history was negative.

Physical examination demonstrated normal blood pressure and clear lungs. Cardiac findings included a normal apical impulse, and normal heart sounds. A late systolic click followed by a grade 2/6 murmur was heard loudest at the apex; it increased in duration and intensity with standing.

An electrocardiogram and chest x-ray were normal, as were CBC counts and serum electrolyte measurements. A two-dimensional echocardiogram was diagnostic of mitral valve prolapse; ventricular size and function were normal. Twenty-four ambulatory electrocardiographic recordings documented frequent (10 to 136 per hour) isolated uniform ventricular premature beats without couplets or other complex forms.

Case Discussion

This case represents one of disabling ventricular arrhythmias not controlled by usual single-drug therapy in a patient without significant structural cardiac disease. Before discussing what was advised in this specific case and what may be advised in other patients with uncontrolled arrhythmias, it is first important to answer several questions.

What is the goal of antiarrhythmic drug therapy? Two general goals of antiarrhythmic drug therapy may be defined. First, in patients with symptoms clearly referable to arrhythmia, therapy may be advised to abolish symptoms. In most cases, the symptoms of syncope or light-headedness result from the hemodynamic consequences of complex ventricular or supraventricular arrhythmias with very high or very low ventricular rates. In others, such as in the patient presented, symptoms are more subjective, and may be limited to palpitations. Whereas in the former case therapy is needed to prevent potentially dangerous episodes, in the latter it may be needed only in the unusual patient in whom symptoms prove disabling and unresponsive to reassurance and other nonpharmacologic approaches.

Second, ventricular arrhythmias may be treated to prevent sudden cardiac death. This is the most common indication, as most patients with serious ectopy are asymptomatic. However, not all arrhythmias are a significant risk to the patient. Table 1, modified from work of Bigger[1] and Anderson,[2] lists electrophysiologic and clinical characteristics of three groups of arrhythmias that pose increasing threat. In general, more complex arrhythmias in patients with more significant left ventricular dysfunction present the greatest risk. These patients require aggressive therapy.[3] In contrast, ectopy of any complexity in subjects without structural heart disease presents little risk.[4] These patients generally do not require therapy to prevent sudden death, though they may need therapy if symptoms, such as palpitations in the case presented, are disabling.

Does antiarrhythmic drug therapy prevent sudden cardiac death? Although drug therapy can prevent symptoms, its ability to prevent sudden cardiac death is less clearly established. Convincing data do demonstrate a reduction in sudden death rate in patients with sustained ventricular tachycardia or fibrillation.[5,6] As shown in Fig. 1, patients on successful antiarrhythmic therapy—

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Presented April 22, 1987.

TABLE 1

FACTORS DETERMINING CLINICAL SIGNIFICANCE OF VENTRICULAR ARRHYTHMIAS

	Benign	Potentially Lethal	Lethal
Risk of SCD	Minimal	Moderate	High
Heart disease	None; Normal EF	CAD, CM, etc.; EF reduced	CAD, CM, etc.; EF<30%
Type of arrhythmia	Normal (VPB <5/hr, 100/day) VPBs, NSVT, VC	VPBs, VC, NSVT	SVT, torsade, NSVT, VF
Hemodynamic consequences	None	None	Present
Treatment	Reassurance; control symptoms	Control symptoms; prevent SCD	Alleviate symptoms; prevent SCD

Key: CAD coronary artery disease; CM cardiomyopathy; EF ejection fraction; NSVT nonsustained ventricular tachycardia; SCD sudden cardiac death; SVT sustained ventricular tachycardia; VC ventricular couplets; VF ventricular fibrillation; VPB ventricular premature beat.

whether judged by invasive or noninvasive methods and criteria—have significantly lower mortality rates than those in whom adequate therapy could not be prescribed.

It is important to emphasize that these studies included only patients with the most complex degrees of ectopy or sudden death. The role of therapy in patients with less complex arrhythmias, including the very large cohort with ectopy or nonsustained ventricular tachycardia after acute myocardial infarction, is not as yet unequivocally defined. National multicenter trials aimed at resolving this question are currently underway.[7]

What is the definition of an uncontrolled arrhythmia? Implicit to any consideration of "uncontrolled" arrhythmias is the definition of what constitutes adequate "control." In general, "control" is achieved when predetermined goals of therapy are met; if these goals are not met, the arrhythmia is "uncontrolled."

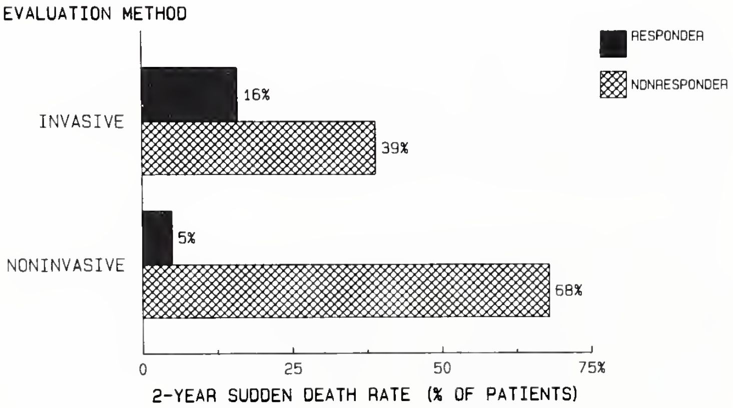

Figure 1. Two-year sudden death rate in patients who respond and who do not respond successfully to antiarrhythmic drug therapy. Data from one study using invasive electrophysiologic methods[6] and from one using ambulatory electrocardiographic monitoring[5] are presented.

Table 2 lists currently considered goals or endpoints of therapy. First, suppression of symptoms may be either a solitary goal of therapy in patients with symptomatic but not malignant ectopy, or an associated goal in subjects with symptomatic and potentially lethal arrhythmias. Because most patients with ectopy requiring therapy are not symptomatic, this therapeutic endpoint is not commonly an isolated goal.

Second, therapy may be given until specified grades of arrhythmias are reduced below a selected frequency on ambulatory electrocardiographic monitoring. Common goals include total elimination of sustained ventricular tachycardia and either complete or near complete (90%) abolition of nonsustained ventricular tachycardia and other repetitive forms of ventricular ectopy. As shown in Fig. 1, control by this method does reduce death rate. Although ambulatory monitoring is routinely available, problems remain in implementing this endpoint, including specification of exact criteria for success, and exclusion of spontaneous variability as being responsible for an apparent drug-induced reduction (or increase) in arrhythmia frequency.[8] It is important to emphasize, however, that not all ectopy need be abolished to achieve excellent clinical results; drug doses to eliminate all or almost all simple ectopy may exceed those needed to treat complex forms,

TABLE 2

ENDPOINTS OF ANTIARRHYTHMIC DRUG THERAPY

Suppression of symptoms
Suppression of arrhythmia by grade and frequency
Suppression of inducibility of complex forms of ventricular ectopy
Maintenance of therapeutic drug levels

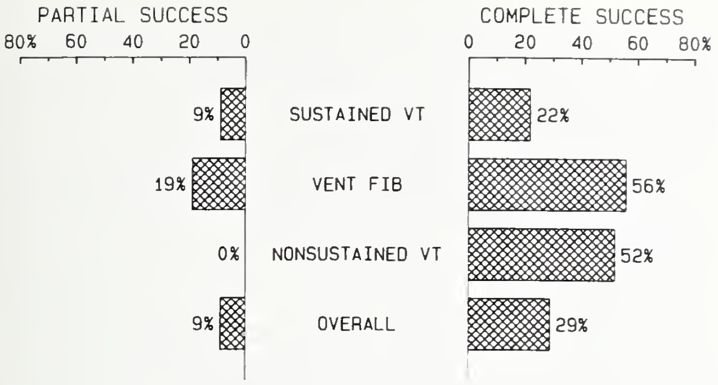

Figure 2. Percentage of patients with sustained or nonsustained ventricular tachycardia, or ventricular fibrillation who could be successfully treated by single or multiple drug therapies. Criteria for "success" were based upon invasive electrophysiologic criteria[11]; "complete" success was defined as initiation of fewer than six repetitive ventricular responses while "partial" success was initiation of six to fifteen repetitive responses in patients with sustained ventricular tachycardia or fibrillation.

and may unnecessarily produce intolerable side effects.[9]

Third, invasive electrophysiologic testing may be used to determine drug efficacy.[10] In patients in whom sustained ventricular tachycardia, clinically relevant ventricular fibrillation, or (perhaps) nonsustained ventricular tachycardia can be induced prior to drug therapy, failure to induce the arrhythmia after drug therapy marks success. Successful therapy does lower the death rate more than unsuccessful therapy[6] (Fig. 1). Although this approach is invasive and more expensive than ambulatory monitoring, it can be applied to patients without high grade ectopy but with previous clinical events such as sudden death or syncope.

Last, maintenance of therapeutic blood levels of one or more agents may be a goal of therapy. Although some data have suggested that achieving only this goal may prevent sudden death,[9] it is best considered as an adjunctive endpoint to be achieved in conjunction with either of the above goals.

Whatever approach or combination of endpoints is used, it is critical to formally determine the goals of therapy *before* therapy is initiated. Only in this way can the need for and the adequacy of therapy be determined.

In how many cases is antiarrhythmic drug therapy "successful"? Although successful antiarrhythmic drug therapy can reduce the incidence of sudden cardiac death in selected patient groups, achieving such results is difficult. This is shown in Fig. 2; the percentage of patients with "complete" or "partial" success for each major class of ventricular arrhythmia is depicted. As can be seen, the success rate—either partial or complete—is relatively low, and varies with the type of arrhythmia; the least complex forms are most treatable.[11] Thus, the current problem of managing the "uncontrolled arrhythmia" is both common and potentially important.

What factors predict or produce "uncontrolled arrhythmias"? Failure of antiarrhythmic drugs to control arrhythmias may result from two groups of causes—"patient failures," related to intrinsic clinical conditions, and "doctor failures," reflecting inappropriate prescription of antiarrhythmic agents.

Table 3 lists clinical factors that predict successful and unsuccessful medical therapy. Parameters associated with failure include cardiac dysfunction, and ischemic heart disease particularly with prior myocardial infarction, ventricular aneurysm, or ongoing ischemia. In contrast, a normal heart predicts success; as noted above, however, most such patients do not require therapy.

In addition, other conditions present in the patient may reduce the chance of success. In particular, hypoxia, hypokalemia, hypomagnesemia, acidosis or alkalosis, and thyrotoxicosis or myxedema all reduce the efficacy of antiarrhythmic drugs. Poor compliance, a common problem enhanced by the high incidence of side effects of virtually all antiarrhythmic drugs, is an additional self-evident problem.

Factors listed in Table 4 are causes for failure

TABLE 3

SELECTED CLINICAL CRITERIA PREDICTING SUCCESSFUL OR UNSUCCESSFUL ("PATIENT FAILURE") ANTIARRHYTHMIC DRUG THERAPY

- Factors predicting success
  - Congestive heart failure
  - Prior myocardial infarction
  - Ongoing myocardial ischemia
  - Ventricular aneurysm
- Factors predicting failure
  - Normal heart
  - Failure to induce arrhythmia by invasive electrophysiologic studies

---

TABLE 4

CAUSES OF UNCONTROLLED ARRHYTHMIAS DUE TO INADEQUATE UTILIZATION OF ANTIARRHYTHMIC DRUGS ("DOCTOR FAILURE")

- Wrong diagnosis
- Inappropriate therapeutic goals
- Inadequate drug dose
- Drug toxicity
- Metabolic abnormalities
- Drug interactions
- Proarrhythmic effects

to control arrhythmias that are due or are amenable to changes in medical therapy, i.e., "doctor failures." First, therapy may have been initiated for an incorrectly diagnosed arrhythmia. Such therapy may not only be ineffective but may be unneeded and potentially dangerous.

Second, the goals of therapy may be poorly defined or inappropriate. For example, attempts to abolish all ectopy rather than just complex forms will result in a significantly higher failure rate and will likely produce significant side effects. In contrast, attempts only to prevent symptoms may not adequately protect against sudden death if reduction in spontaneous or induced complex ectopy is not also sought.

Third, the chosen drug dose may be either inadequate, either in amount or in dosing frequency, or excessive. In the former case, drug "failure" may reasonably be expected, and in the latter, the excessive dose may produce untoward electrophysiologic consequences that may produce new or aggravate preexisting arrhythmias. To detect these problems, assay of blood levels of all agents is important, but it must be recognized that an accurate diagnosis of drug toxicity or inadequate dosing depends upon a correlation of clinical and electrocardiologic parameters with serum levels, and not upon drug levels alone.

Fourth, correctable electrolyte or respiratory or metabolic abnormalities may aggravate ventricular ectopy or may impede normal drug action. For example, hypokalemia increases ectopy while reducing effectiveness of many agents. Hypoxia and hypocapnea likewise aggravate preexistent ectopy.

Concomitant drug therapy may likewise prevent adequate control. Such interaction may occur by any of several mechanisms, including a direct aggravation of underlying arrhythmias (e.g., by a methylxanthine or drug intoxication), production of electrolyte or metabolic imbalance (e.g., diuretic induced hypokalemia), alteration of drug metabolic rates (e.g., reduced lidocaine clearance by propranolol), and competitive binding (e.g., increased digitalis levels produced by quinidine).

Finally, the drug given to reduce ectopy may itself aggravate ectopy.[11] This proarrhythmic effect may result from indirect mechanisms such as aggravating congestive heart failure or by direct electrophysiologic actions. The latter include depressing cardiac conduction or automaticity to permit reentrant or escape rhythms to emerge. The clinically paradoxic outcome occurs in approximately 10% of patients[12] and may be more common in those with more severe ventricular dysfunction[13] and more complex ectopy, i.e., those that most urgently require effective antiarrhythmic drug therapy.

After consideration of all of the above factors, what can be done to achieve control in the patient with a presently "uncontrolled arrhythmia"? (Note the difference between "uncontrolled" and "uncontrollable.") Possible approaches are listed in Table 5.

First, problems listed in Table 4 should be corrected. Approaches include reassessment of arrhythmia diagnosis, reassessment of need for and the goals of drug therapy, determination of blood drug levels and assessment of adequacy of drug dose, correction of underlying electrolyte or metabolic imbalances, and reevaluation of concomitant drug therapy for other related or unrelated diseases to exclude deleterious pharmaco-

TABLE 5

APPROACHES TO THE PATIENT WITH AN UNCONTROLLED ARRHYTHMIA

- Correction of iatrogenic factors
- Correction of reversible disease factors
- Alterations in antiarrhythmic drug therapy
    - Dose reduction
    - Supratherapeutic doses
    - Alternate agents
    - Drug combinations
- Nonpharmacologic therapy
    - Antitachycardia pacemakers
    - Implantable cardioverters/defibrillators
    - Endocardial excision

logic or pharmacokinetic interactions.

Second, clinical abnormalities related to the patient's underlying diseases should be corrected. This may include increasing antianginal agents to ameliorate continuing myocardial ischemia, altering digitalis or diuretic therapy to reduce arrhythmogenic myocardial stretch, or improving control of hypertension, hypoxia, or thyrotoxicosis.

Only after the prior concerns have been resolved should alterations in antiarrhythmic drug therapy be considered. First, to exclude proarrhythmic effects that mimic intrinsic lack of control, consideration should be given to stopping or, at least, reducing doses of all agents. This must however be done under close monitoring to detect emergence of more severe, life-threatening arrhythmias that may not have been previously observed.[14]

Second, increasing drug dosages to produce serum levels beyond the usually recommended range may improve control. These ranges are beyond those that produce "control" in a population sample; individual patients may require substantially higher levels, and may not develop toxicity.

Third, one agent may be switched to another. Even within the same pharmacologic group of drugs, patients may respond to one but not another agent. For example, quinidine may be effective when neither procainamide nor disopyramide achieves adequate control.

Fourth, drugs may be given in combination. Two general approaches may be used to choose agents to combine. First, two similar agents may be given at lower doses to achieve control but without the side effects produced by full doses of either agent. For example, moderate doses of quinidine and procainamide may achieve control when either one given alone causes intolerable complaints at doses too low to be effective.[15]

Alternatively, two agents with differing pharmacologic properties may be selected. This combination may reduce side effects (e.g., adding mexilitine to quinidine may reduce the drug-induced prolonged QT interval), or may directly improve efficacy. The latter may be accomplished by selecting agents with very different modes of action, for example, adding a beta-adrenergic antagonist or a drug that effects predominantly repolarization (e.g., amiodarone) to an agent predominantly affecting sodium channels. Guidelines have been recently detailed by Greenspan et al.[16]

Finally, when none of these approaches produces satisfactory results, nonpharmacologic methods should be considered. These include implantation of antitachycardia pacemakers or cardioverter/defibrillators[17-19] and direct surgical resection of the arrhythmogenic tissue.[19,20] Both antitachycardia pacemakers and implantable cardioverters/defibrillators contain circuits and logic to diagnose ventricular tachycardia or fibrillation. Tachycardias may be broken by introducing appropriately timed extrastimuli to break the reentrant circuit—just as may be done in the clinical electrophysiology laboratory. Alternately, both sustained ventricular tachycardia and fibrillation may be successfully converted to sinus rhythm by delivery of one or more 20-30 joule shocks through implanted epicardial and intracavitary electrodes. Although these devices are new and expensive and do require a thoracotomy to implant, early experience suggests a major role for future devices in clinical management.[17-19]

Direct extirpation of damaged tissue responsible for initiation of ventricular arrhythmias has also proven to be feasible and, in many cases, highly successful. The most common approach involves resection of grossly aneurysmal myocardium plus a margin of subendocardial tissue to include sites of tachycardia initiation as determined by intraoperative electrophysiologic mapping.[19,20]

All of these latter approaches require complex and sophisticated preoperative evaluations. This is true for pacemaker implants (intended antitachycardia modes may produce acceleration of the rhythm with degeneration to fibrillation) as well as for operative therapies.

I hope this review has identified key approaches to diagnosis and management of complex but uncontrolled arrhythmias. Specific guidelines may be found in the cited references. It must be repeatedly emphasized that none of the approaches should be considered without a careful clinical evaluation and reevaluation to assess the need for therapy, the goals of therapy, and the presence of correctable factors. New pharmacologic agents as well as electronic and surgical approaches will clearly expand the possibilities that may reduce the presently high incidence of sudden cardiac death, but will not replace basic clinical judgment.

The case presented illustrates this concept. Careful and prolonged interviewing suggested that most palpitations and disability occurred after relatively sleepless nights. Prescription of a hypnotic agent (flurazepam, 30 mg at bedtime) improved rest, reduced ectopic frequency, and markedly improved the disabling symptoms. No further antiarrhythmic drug therapy was needed.

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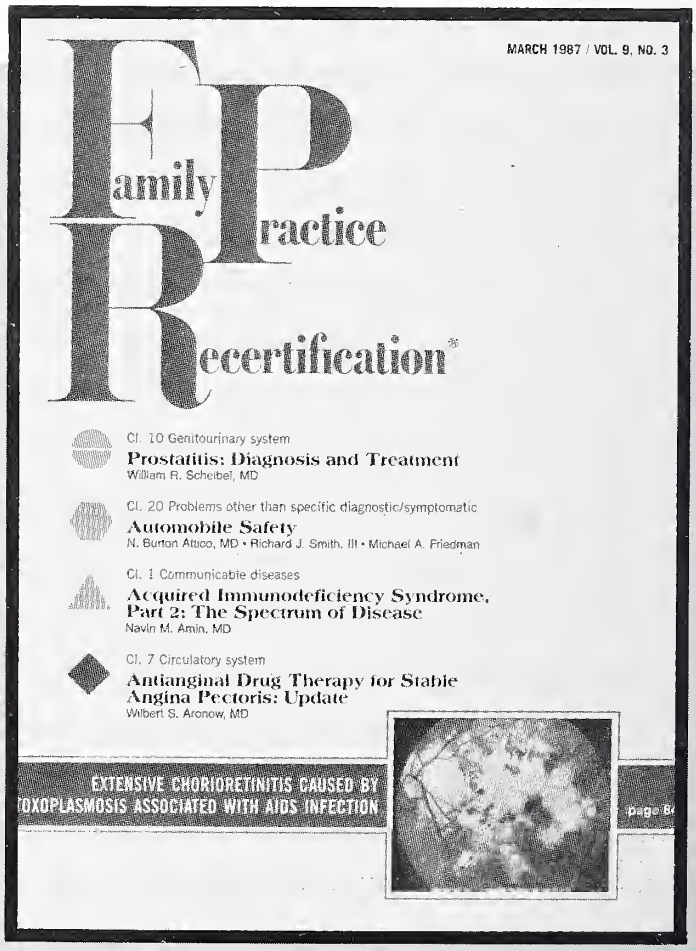

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A Case of Rhabdomyolysis

Case Report

A 29-year-old Hispanic man with a history of heavy alcohol and cocaine abuse was admitted to Vanderbilt Hospital with acute psychosis. He had been well until the day before admission, when he had drunk 12 beers and used intranasal cocaine. Shortly thereafter, he became violent and irrational, and was brought to Vanderbilt Hospital confused and combative and requiring restraint. In the emergency room, his mental status improved, but when his catheterized urine was noted to be tea colored, he was admitted for observation.

His physical examination was negative except for drowsiness; he had no muscle tenderness or swelling. Laboratory examination showed a metabolic acidosis, with a serum pH of 7.26 (normal 7.35 to 7.45), P_{CO_2} of 26 mm Hg (normal 35 to 45) and a serum bicarbonate of 17 mmol/L (normal 22 to 30). His electrolytes were normal and his creatinine was 1.0 mg/dl (normal 0.6 to 1.2). Other values included an LDH of 1,330 IU/L (normal 125 to 250), an SGOT of 410 IU/L (normal 4 to 40), a total bilirubin of 0.4 mg/dl (normal 0.2 to 1.2) and a CPK of 21,000 IU/L (normal 30 to 210). CBC count, platelet count, and coagulation studies were normal. His urine was brown and dipstick positive for albumin, acetone, and a trace of blood, but no red cells were seen. Urine for myoglobin was negative.

It was thought that the patient had rhabdomyolysis, and he was vigorously hydrated to maintain a high urine output. His serum CPK peaked at 29,000 IU/L on day 3 of hospitalization, and subsequently decreased, his creatinine remained normal, and his metabolic acidosis resolved.

Comment

Rhabdomyolysis is the clinical and laboratory syndrome resulting from skeletal muscle injury; translated, it means "skeletal muscle dissolution." The usual clinical syndrome emphasizes muscle pain, swelling, and weakness, with dark urine, although some or none of these findings may be present.[1] The diagnosis is confirmed biochemically by measuring substances released from damaged muscle tissue, which include CPK, LDH, SGOT, aldolase, myoglobin, potassium, phosphate and purine metabolites, CPK being the most sensitive marker.

Rhabdomyolysis may or may not result in detectable myoglobinuria, depending on the amount of myoglobin released into the plasma, the glomerular filtration rate, and the urine concentration. Since myoglobin is cleared in one to six hours by urinary excretion, it is often not detected.[1]

Gabow et al[1] studied the records of 87 patients with rhabdomyolysis and found alcoholism to be the most common etiologic factor. Compression of muscle associated with lying immobile for long periods was second. Multiple factors caused the condition in 59%, including the strenuous exercise of military recruits at boot camp.[2,3]

One of the more common and serious consequences of rhabdomyolysis is renal failure. Its frequency is unknown, but may be as high as 33%.[1] Its cause in this situation is also unknown, as myoglobin itself does not seem to induce it.[4] Further, the risk of renal failure does not correlate with peak CPK values.[1]

Treatment of rhabdomyolysis is primarily supportive and aimed at maintaining a high urine output and treating any metabolic abormalities (i.e., disseminated intravascular clotting, hyperkalemia, and metabolic acidosis) that may occur.

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Meningoencephalitis in a Heart Transplant Recipient

Case Report

A 49-year-old white man who had received a cardiac transplant six months earlier was admitted to Vanderbilt Hospital with fever, confusion, and meningismus. Congestive heart failure began six years prior to transplant, progressing to end stage disease with a dilated cardiomyopathy of unclear etiology. The patient subsequently received an orthotopic cardiac transplant complicated postoperatively by *Staphylococcus epidermidis* sepsis, mild rejection reaction, and hepatic dysfunction. He subsequently did well on a regimen of cyclosporine, azathioprine, and prednisone until the day prior to admission, when he developed fever, headache and ma-

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Prepared by Jason D. Morrow, M.D., Hugh J. Morgan chief medical resident, Vanderbilt Medical Center, Nashville.

laise; he subsequently became confused and was admitted to Vanderbilt University Hospital.

Physical examination showed a well-developed white man unresponsive to commands. His temperature was 103.6°F rectally, pulse 120/min, and blood pressure 140/90 mm Hg. Except for meningismus, the remainder of his physical examination was negative.

Laboratory examination showed a peripheral WBC count of 13,600/cu mm (normal 4,000 to 10,000) with 10% band forms, 88% polymorphonuclear cells, and 2% lymphocytes on the differential. Other laboratory results were normal, as was CT scan of the head with and without contrast. A lumbar puncture revealed an opening pressure of 350 mm H_2O, with a protein of 120 mg/dl (normal 15 to 45), glucose 44 mg/dl (normal 45 to 75), and cell count of 300 WBCs/cu mm, of which 73% were neutrophils and 27% were monocytes; there were 710 RBCs/cu mm. Gram stain and India ink preparation were negative for organisms and the cryptococcal antigen was negative. The patient was placed on cefotaxime 2 gm IV every four hours, ampicillin 2 gm IV every four hours, and gentamicin 100 mg IV every eight hours. Subsequent blood and CSF cultures grew *Listeria monocytogenes*. Once culture results were known, the patient was treated with ampicillin alone for three weeks and recovered completely.

Comment

Listeria monocytogenes is a Gram-positive rod shaped motile aerobic bacterium associated with a variety of infections. In the immunosuppressed patient, it most often causes sepsis and meningoencephalitis,[1] the latter when bacteremia leads to seeding of the meninges and brain. The presentation may be subtle or fulminant. In some studies of immunosuppressed patients, it is the most common cause of meningitis.[2] Further, it is being recognized with increased frequency as a cause of serious infections in renal transplant recipients, and has been reported as a cause of meningitis in heart transplant patients.[3,4] Host factors that appear to predispose to infection are T cell-mononuclear cell defects.

CSF findings in *Listeria* meningoencephalitis are variable, with WBC counts ranging from 6,000 to 12,000/cu mm. CSF protein may also vary, and CSF glucose values can be low or normal.[1] The Gram stain of the CSF is often negative, but the organism will grow easily on standard media.

The therapy of choice is penicillin or ampicillin, although resistance to both antibiotics has been shown. Some studies note synergism with these agents and aminoglycosides. Chloramephenicol or cotrimoxazole may be alternative agents for treatment, but data are sparse.[1]

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Loss Prevention Case of the Month

Come to My Office in the Morning

J. KELLEY AVERY, M.D.

With very few exceptions, every physician who reads this column will have made that statement many times during the course of his practice. Most of the time such a statement is entirely justified, and is extremely good advice, but once in a while an event occurs to make us realize that "come to my office in the morning" is a piece of advice that must be given with due regard to the circumstances.

Case Report

A 58-year-old man had been treated in the hospital for an acute myocardial infarction complicated by severe, persistent pain and some congestive heart failure. His initial period of hospitalization was something slightly less than two weeks. He became pain-free after four or five days in the hospital, and although he developed significant pulmonary congestion, on proper diuretics and low-salt intake his lungs cleared; at the time of discharge he was pain-free and breathing easily.

On leaving the hospital he was given instructions about medications, which included nitroglycerin and a diuretic to be taken daily. A graduated program of exercise was prescribed, and he was given an appointment to see his cardiologist two weeks from the time of discharge.

During the six days following discharge, there were four telephone calls from the patient, each time complaining of chest pain, and each time he talked with a resident who had seen him while he was in the hospital. He was advised to take his nitroglycerin regularly, was reassured, and reminded of his appointment with the cardiologist. Nine days after discharge, on a very busy night in the emergency room (ER), he appeared at 3 AM with severe chest pain, marked diaphoresis, and extreme shortness of breath. The hospital was very crowded, and there were no available beds in the coronary care unit (CCU). He was observed for about six hours in the ER and given morphine, IV diuretics, and oxygen support with marked improvement. His chest pain subsided, and with oxygen he was no longer short of breath. After the period of observation, he was allowed to go home and was told to "see his cardiologist in the morning." He returned to the ER two hours later in severe congestive heart failure, experienced cardiopulmonary arrest in the ER, and died two days later.

After a lawsuit was filed, the investigation revealed that this patient's four calls to his cardiologist had been handled by two different residents. Although they communicated with the cardiologist, there was no effort on their part or the part of the specialist to have the patient speak with the cardiologist himself. Investigation disclosed that on the night when the patient went to the ER with chest pain and shortness of breath, the cardiologist approved by telephone the resident's decision to allow the patient to go home after the six-hour observation period, during which he responded to treatment. Litigation resulted in a large settlement.

Loss Prevention Comments

The lawsuit charged the residents and the cardiologist with negligence in the care of the patient. One of the most damaging facts uncovered in the investigation, and one among others that led to the large settlement, was that the "crowded hospital" somehow influenced the decision by the resident and the cardiologist to allow this man to return to his home rather than keep him in the hospital.

Conditions other than those presented by the patient frequently influence our decisions relative to the patient's care; in this very tragic case, it was a "crowded hospital," "the unavailability of a bed in the CCU," and a genuine belief that the patient would be better off at home than he would have been in an unmonitored bed on a regular medical floor that led to his being allowed to return home. Sometimes, when there are no financial impediments to the care of a patient, we might do things differently than we would if the patient were going to be unable to pay his hospital bill. When we practice in an area remote from a tertiary care hospital, we might well "wait" for a patient to stabilize, but when given the same patient in a tertiary care setting our management would be more aggressive. Within limits, these variations in the management of patients under differing circumstances can be fully justified. However, when there is a bad result that can be linked to decisions we make because of circumstances other than those inherent in the condition of the patient, we put ourselves in a position that may be legally hard to defend if litigation results. In this case, it is entirely probable that this patient's outcome would have been the same even if he had been retained in the hospital, but it could also be argued the other way.

Dr. Avery is chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood, and medical director for Ambulatory/Outpatient Services, St. Thomas Hospital, Nashville.

Birth Certificate Changes

PAULA M. TAYLOR, M.S.

Although births have been registered in all Tennessee counties since 1914, the form of the certificate has changed through the years. Few documents have the legal significance of the birth certificate. As our society has become more complex, persons are required more frequently to provide proof of age, parentage, and citizenship; thus, the birth certificate is more important now than ever before.

Because of their dual purpose—legal and statistical—vital records maintain a unique role in the public health field. Vital statistics obtained from the record are recognized as fundamental to the planning and administration of public health programs, both governmental and nongovernmental.

The vital records system in the United States is unlike that in most other countries because the U.S. system is decentralized and cooperative. Responsiblity for registration of vital events belongs to the individual states and seven other independent registration areas. In order to achieve national uniformity and thus contribute to a national vital statistics system, these 57 registration areas strive to adopt or otherwise conform to nationally recommended reporting forms (termed U.S. Standard Certificates), definitions, and laws and regulations.

In Tennessee, the registration of vital events (births, defects, fetal deaths, induced terminations of pregnancy, marriages, and divorces) is the responsibility of the Tennessee Department of Health and Environment, but the Department depends on practicing physicians, medical records staffs, county clerks, and others to provide the information.

Approximately every ten years, the format and content of the vital records in this country are evaluated by a panel of national experts to assure that the requested information reflects current technology and societal attitudes and meets the needs of data users. This periodic evaluation has recently been completed, and recommendations for changes to the certificates have been transmitted to the vital records offices in all states.

Representatives of the American College of Obstetricians and Gynecologists, the American Hospital Association, the Association for Maternal and Child Health and Crippled Children's Programs, state health departments, and two researchers (one in neonatology and one in demography) comprised the panel that evaluated the Standard Birth Certificate. The panel's recommendations were intended to reflect concern for issues likely to remain important over the next decade—environmental health, occupational health services, control of noninfectious diseases, and changes in the health care delivery system.

The newly recommended national Standard Birth Certificate (Figure) reflects some significant changes from the certificate now in use. The first change is that medical items on the certificate (complications of pregnancy, concurrent illnesses or conditions affecting the pregnancy, and congenital malformations of the child) are being reformatted from open-ended responses to specific check-box responses. This approach is expected to result in more complete, usable, and uniform data.

The second major recommendation is the addition of three new items, in check-box format, to obtain information on obstetric procedures, method of delivery, and abnormal conditions of the newborn. These items will attempt to measure the use of certain technologies in obstetrics as well as the incidence of specific abnormal newborn conditions.

The third major change involved replacing the existing open-ended question about the ethnic origin of the parents with a specific inquiry about

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From the Center for Health Statistics, Tennessee Department of Health and Environment, Nashville.

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U.S. STANDARD
CERTIFICATE OF LIVE BIRTH

LOCAL FILE NUMBER

BIRTH NUMBER

CHILD

1. CHILD'S NAME *(First, Middle, Last)*

2. DATE OF BIRTH *(Month, Day, Year)*

3. TIME OF BIRTH
M

4. SEX

5. CITY, TOWN, OR LOCATION OF BIRTH

6. COUNTY OF BIRTH

7. PLACE OF BIRTH: ☐ Hospital ☐ Freestanding Birthing Center
☐ Clinic/Doctor's Office ☐ Residence
☐ Other *(Specify)* ____

8. FACILITY NAME *(If not institution, give street and number)*

CERTIFIER/ ATTENDANT

9. **I certify that this child was born alive at the place and time and on the date stated.**
Signature ▶

10. DATE SIGNED *(Month, Day, Year)*

11. ATTENDANT'S NAME AND TITLE *(If other than certifier) (Type/Print)*
Name ____
☐ M.D. ☐ D.O. ☐ C.N.M. ☐ Other Midwife
☐ Other *(Specify)* ____

12. CERTIFIER'S NAME AND TITLE *(Type/Print)*
Name ____
☐ M.D. ☐ D.O. ☐ Hospital Admin. ☐ C.N.M. ☐ Other Midwife
☐ Other *(Specify)* ____

13. ATTENDANT'S MAILING ADDRESS *(Street and Number or Rural Route Number, City or Town, State, Zip Code)*

DEATH UNDER ONE YEAR OF AGE
Enter State File Number of death certificate for this child

14. REGISTRAR'S SIGNATURE
▶

15. DATE FILED BY REGISTRAR *(Month, Day, Year)*

MOTHER

16a. MOTHER'S NAME *(First, Middle, Last)*

16b. MAIDEN SURNAME

17. DATE OF BIRTH *(Month, Day, Year)*

18. BIRTHPLACE *(State or Foreign Country)*

19a. RESIDENCE—STATE

19b. COUNTY

19c. CITY, TOWN, OR LOCATION

19d. STREET AND NUMBER

19e. INSIDE CITY LIMITS? *(Yes or no)*

20. MOTHER'S MAILING ADDRESS *(If same as residence, enter Zip Code only)*

FATHER

21. FATHER'S NAME *(First, Middle, Last)*

22. DATE OF BIRTH *(Month, Day, Year)*

23. BIRTHPLACE *(State or Foreign Country)*

INFORMANT

24. **I certify that the personal information provided on this certificate is correct to the best of my knowledge and belief.**
Signature of Parent or Other Informant ▶

INFORMATION FOR MEDICAL AND HEALTH USE ONLY

	25. OF HISPANIC ORIGIN? (Specify No or Yes—If yes, specify Cuban, Mexican, Puerto Rican, etc.)	26. RACE—American Indian, Black, White, etc. *(Specify below)*	27. EDUCATION *(Specify only highest grade completed)* Elementary/Secondary (0-12)	College (1-4 or 5+)
MOTHER	25a. ☐ No ☐ Yes Specify:	26a.	27a.	
FATHER	25b. ☐ No ☐ Yes Specify:	26b.	27b.	

28. PREGNANCY HISTORY *(Complete each section)*

LIVE BIRTHS *(Do not include this child)*		OTHER TERMINATIONS *(Spontaneous and induced at any time after conception)*
28a. Now Living Number ____ ☐ None	28b. Now Dead Number ____ ☐ None	28d. Number ____ ☐ None
28c. DATE OF LAST LIVE BIRTH *(Month, Year)*		28e. DATE OF LAST OTHER TERMINATION *(Month, Year)*

29. MOTHER MARRIED? (At birth, conception, or any time between) *(Yes or no)*

30. DATE LAST NORMAL MENSES BEGAN *(Month, Day, Year)*

31. MONTH OF PREGNANCY PRENATAL CARE BEGAN—First, Second, Third, etc. *(Specify)*

32. PRENATAL VISITS—Total Number *(If none, so state)*

33. BIRTH WEIGHT *(Specify unit)*

34. CLINICAL ESTIMATE OF GESTATION *(Weeks)*

35a. PLURALITY—Single, Twin, Triplet, etc. *(Specify)*

35b. IF NOT SINGLE BIRTH—Born First, Second, Third, etc. *(Specify)*

MULTIPLE BIRTHS
Enter State File Number for Mate(s)
LIVE BIRTH(S)

FETAL DEATH(S)

36. APGAR SCORE
36a. 1 Minute
36b. 5 Minutes

37a. MOTHER TRANSFERRED PRIOR TO DELIVERY? ☐ No ☐ Yes If Yes, enter name of facility transferred from:

37b. INFANT TRANSFERRED? ☐ No ☐ Yes If Yes, enter name of facility transferred to:

38a. MEDICAL RISK FACTORS FOR THIS PREGNANCY *(Check all that apply)*

Anemia (Hct. <30/Hgb. <10)	01	☐
Cardiac disease	02	☐
Acute or chronic lung disease	03	☐
Diabetes	04	☐
Genital herpes	05	☐
Hydramnios/Oligohydramnios	06	☐
Hemoglobinopathy	07	☐
Hypertension, chronic	08	☐
Hypertension, pregnancy-associated	09	☐
Eclampsia	10	☐
Incompetent cervix	11	☐
Previous infant 4000+ grams	12	☐
Previous preterm or small-for-gestational-age infant	13	☐
Renal disease	14	☐
Rh sensitization	15	☐
Uterine bleeding	16	☐
None	00	☐
Other ____ *(Specify)*	17	☐

38b. OTHER RISK FACTORS FOR THIS PREGNANCY *(Complete all items)*

Tobacco use during pregnancy .... Yes ☐ No ☐
Average number cigarettes per day ____
Alcohol use during pregnancy .... Yes ☐ No ☐
Average number drinks per week ____
Weight gained during pregnancy ____ lbs.

39. OBSTETRIC PROCEDURES *(Check all that apply)*

Amniocentesis	01	☐
Electronic fetal monitoring	02	☐
Induction of labor	03	☐
Stimulation of labor	04	☐
Tocolysis	05	☐
Ultrasound	06	☐
None	00	☐
Other ____ *(Specify)*	07	☐

40. COMPLICATIONS OF LABOR AND/OR DELIVERY *(Check all that apply)*

Febrile (>100°F. or 38°C.)	01	☐
Meconium, moderate/heavy	02	☐
Premature rupture of membrane (>12 hours)	03	☐
Abruptio placenta	04	☐
Placenta previa	05	☐
Other excessive bleeding	06	☐
Seizures during labor	07	☐
Precipitous labor (<3 hours)	08	☐
Prolonged labor (>20 hours)	09	☐
Dysfunctional labor	10	☐
Breech/Malpresentation	11	☐
Cephalopelvic disproportion	12	☐
Cord prolapse	13	☐
Anesthetic complications	14	☐
Fetal distress	15	☐
None	00	☐
Other ____ *(Specify)*	16	☐

41. METHOD OF DELIVERY *(Check all that apply)*

Vaginal	01	☐
Vaginal birth after previous C-section	02	☐
Primary C-section	03	☐
Repeat C-section	04	☐
Forceps	05	☐
Vacuum	06	☐

42. ABNORMAL CONDITIONS OF THE NEWBORN *(Check all that apply)*

Anemia (Hct. <39/Hgb. <13)	01	☐
Birth injury	02	☐
Fetal alcohol syndrome	03	☐
Hyaline membrane disease/RDS	04	☐
Meconium aspiration syndrome	05	☐
Assisted ventilation <30 min	06	☐
Assisted ventilation ≥30 min	07	☐
Seizures	08	☐
None	00	☐
Other ____ *(Specify)*	09	☐

43. CONGENITAL ANOMALIES OF CHILD *(Check all that apply)*

Anencephalus	01	☐
Spina bifida/Meningocele	02	☐
Hydrocephalus	03	☐
Microcephalus	04	☐
Other central nervous system anomalies *(Specify)* ____	05	☐
Heart malformations	06	☐
Other circulatory/respiratory anomalies *(Specify)* ____	07	☐
Rectal atresia/stenosis	08	☐
Tracheo-esophageal fistula/Esophageal atresia	09	☐
Omphalocele/Gastroschisis	10	☐
Other gastrointestinal anomalies *(Specify)* ____	11	☐
Malformed genitalia	12	☐
Renal agenesis	13	☐
Other urogenital anomalies *(Specify)* ____	14	☐
Cleft lip/palate	15	☐
Polydactyly/Syndactyly/Adactyly	16	☐
Club foot	17	☐
Diaphragmatic hernia	18	☐
Other musculoskeletal/integumental anomalies *(Specify)* ____	19	☐
Down's syndrome	20	☐
Other chromosomal anomalies *(Specify)* ____	21	☐
None	00	☐
Other ____ *(Specify)*	22	☐

DEPARTMENT OF HEALTH AND HUMAN SERVICES – PUBLIC HEALTH SERVICE – NATIONAL CENTER FOR HEALTH STATISTICS – 1989 REVISION

Hispanic origin. (This does not affect the question concerning race of the parents, which will remain in its present format.) This recommendation resulted from the great interest in the health status and growth of the Hispanic population, which is now the fastest growing ethnic group in this country.

Last, but most obvious, is the change in size from the current 7½ × 9-inch form to legal size (8½ × 14 inch). This change was necessitated not by the addition of new items but rather by the space required for check-box-style responses.

The Tennessee Department of Health and Environment has begun to assess these national recommendations. Before finalizing the design of a new Tennessee birth certificate, department staff will meet with physicians, medical records personnel, and data users to discuss ways to incorporate Tennessee needs into the new form. Since the Tennessee Medical Association will be asked to assist in the design of the new birth certificate, physicians who have recommendations for changes should transmit them to the TMA office or contact Paula M. Taylor, Director, Center for Health Statistics, C2-242 Cordell Hull Building, Nashville, TN 37219 or call Mrs. Taylor at 741-1954 (in Nashville) or toll free (800) 547-3558 (in Tennessee).

Use of this new birth certificate is not scheduled to begin in Tennessee until Jan. 1, 1989. This should provide ample time for input from physicians across Tennessee.

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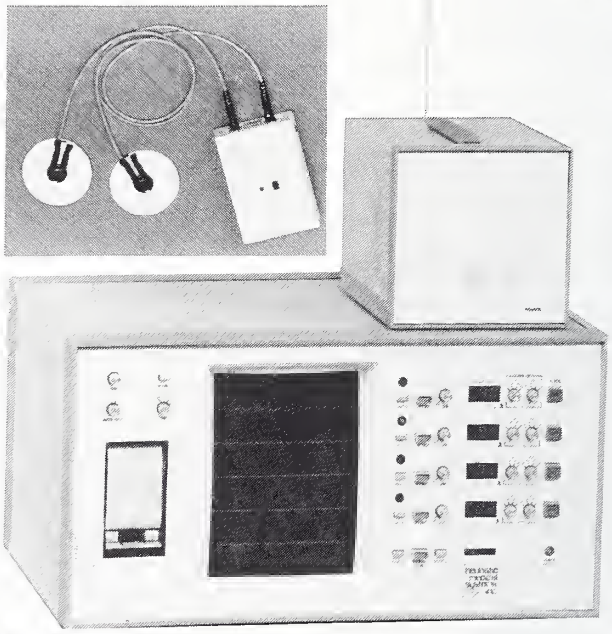

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James T. Galyon

Professionalism

It seems that physicians are having increasing difficulty maintaining their professionalism. The environment in which we are forced to practice increasingly forces us to act more as merchants or purveyors of a service than a group of learned professionals dedicated to serving the public.

As professionals, we correctly assumed that no one but another physician could adequately evaluate our practices and encourage our colleagues to practice ethical medicine. The public appreciated this, and thus far believes that the AMA and its component societies oversee doctors' ethical practices. We know, however, that the FTC and the Supreme Court have virtually emasculated peer review and ethics committees.

Now market forces are forcing physicians farther away from professionalism. Both federal and medical sources have referred to us as providers and as the health care industry so long and so often that even we are beginning to see ourselves in that light. The FTC has insisted so strongly on competition in the marketplace that we have become medical merchants seeking better marketing ploys, joint ventures with non-professionals, and medical care offered at advertised discounts to various groups (PPO members), just like a department store.

Now along comes one more challenge to our professional image. That challenge is physician drug dispensing. We have always had the right to dispense drugs, and have individually decided about doing it. Now the FTC on the one hand is encouraging and even demanding competition at all levels, including drug dispensing, while on the other hand Congress, in response to some pharmacists, is trying to prohibit physicians from dispensing drugs when the service makes a profit for the doctor.

Where does all of this lead—drug dispensing, advertising, marketing, joint venturing with non-professionals? If we are going to continue to enjoy the public's respect, or in some cases to regain lost respect, then we must scrupulously evaluate all of our activities. We need public support to restrain government and market forces that would seriously damage our ability to practice as we would like. We are few in number, and we must maintain our image to have any political clout. Every act that can be interpreted as non-professional will be used to reduce us to an industry, not a profession, and to be regulated like a public utility. The litmus test is simple: if your choice serves the public, your patients, then it is professionalism; if your actions serve you more than your patient, then it is merchandising of medical care. The choice is yours.

James T. Galyon, M.D.

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OCTOBER, 1987

editorials

On Playing Drugstore

Since surfacing a couple of years ago, the issue of dispensing of drugs by physicians, apparently introduced by the wholesale drug packaging industry, has generated a lot of conversation and not a little controversy. It is clearly turf warfare, and, as usual, economics is at the bottom of the quarrel.

Historically, doctors have always had the freedom to dispense medications, and until recently did so routinely as a part of their practice. How much they relegated to the pharmacy was a matter of convenience and economy. As it finally became uneconomical to dispense drugs, the practice dwindled and ultimately for practical purposes disappeared.

With the growth of the wholesale drug industry and the accessibility of medications at vastly discounted prices, it has once again become feasible for doctors to do their own dispensing. Several resolutions before the AMA House of Delegates at the Annual Meeting last June called for the AMA to support physician drug dispensing with greater or less enthusiasm and aid; what was ultimately adopted was a substitute resolution that the AMA support the physician's right to dispense drugs and devices when it is in the best interests of the patient and consistent with AMA's ethical guidelines.

Though there is obviously some room for conflict of interest here, the statutory limitation before the Congress being pressed by the pharmacists' lobby is just another attempted incursion into our prerogatives. A doctor should be able to dispense to his patient whatever he thinks the patient needs if he wants to do it, providing he does not profiteer from it, and advises the patient that the medication or device could be obtained elsewhere. To penalize the doctors just to feather the nest of another group would seem to me an unconstitutional intrusion by government into an area where it has no business, though of course that would not be the first time that has happened.

I cannot imagine that the amount of dispensing doctors would do would constitute any huge threat to the pharmacists. Out of self-preservation if nothing else, few doctors are going to stock a great, extensive inventory. It is expensive, particularly since most items will be used infrequently, and will become outdated, which will constitute a drain on finances. I should think that rather than being the immense bonanza predicted by the pharmaceutical marketers it would instead be an expensive pain in the nether regions. Most doctors will doubtless stock a few drugs they use frequently and nothing more. Even if they start out big, I predict most will soon regroup and let the pharmacists have it, which most of us will be content to do anyway.

It is my considered opinion that this is a tempest in a teapot, and for once in its busybody existence, Congress should resist the urge to stick its nose where it has no business and let the marketplace operate. It is up to the medical profession to be sure its members behave ethically, but of course that other super-busybody, the FDA, likely will not let us. If they are fair—but they aren't, so I won't bother saying what if.

J.B.T.

Sue the Bastard!

For every problem there is an obvious solution that is easy, simple, and wrong. Such an approach is the one taken by the federal bureaucracy and many in the Congress, notably Senator Kennedy, toward the present medical liability crisis. It holds that if only doctors were better educated in current procedures there would not be so much malpractice, there would not then be so many lawsuits, and the premiums would consequently not be so high. Some medical educators in high places have cried, "Yea, verily," and have proffered as the panacea mandatory CME, a solution adopted by many state legislatures, and even mandatory examinations to document learning. When professional liability carriers respond that such a solution mistakes the problem, they are accused of distorting the facts to serve their own nefarious, greedy ends.

In fact, with few exceptions, "malpractice" suits have almost nothing to do with malpractice, and they are for the most part not against bad doctors. A large percentage of the litigation involves skillful practitioners with bad manners, or doctors who have office staff with bad manners. Most of the rest of the suits are for predictable bad results for which someone (usually a hungry plaintiff's attorney) is seeking indemnification. What is needed most is not continuing medical education, but education in etiquette and the consequences of lack of it. "Miss Manners" could do a lot for us.

A relatively brief term of service on SVMIC's Claims Review Committee has indicated to me that patients often sue for no better reason than that they are mad at the doctor; this pique is exposed to some third party, ultimately a lawyer with visions of dollars dancing in his head, who says, "Sue the bastard" (though his language may be either more or less couth). And so it happens. What the patient is usually mad about is that the doctor did not show what the patient considered appropriate concern about the perceived gravity of the situation. Doctors need to learn that it does not really matter whether or not the situation is grave. If the patient (or, sometimes more to the point, the patient's family) thinks it is, it is. A cavalier attitude can make a doctor into a case. Though the tort will doubtless be presented as less than ideal results, it is in fact mental anguish, or simply venting spleen.

I had a doctor friend say to me once that as a patient he was talked to at length, but never communicated with, by his doctor about his condition, and that he therefore never gave *informed* consent. He was, he said, under the circumstances incapable of being informed. Furthermore, no one in his family was acting out of informed consent. Even with our best efforts, communication is difficult and failure is easy and frequent. With the almost continuous publicity over medical advances, many of them still in the experimental stages, public expectations are seldom realistic. When a bad result is anticipated, the word may not get through to the patient and his family, and when the inevitable occurs, it may be assumed to be due to negligence or lack of skill, and a lawsuit follows. Sometimes the motivation is purely economic, a search for indemnification from the deep pocket. Such liability is incurred by the best, not the worst, doctors, since only the best will tackle the more hopeless cases.

Of all the cases to come before the Claims Review Committee, few show deviations from accepted standards of care, and most of the cases where settlement is recommended suffer mostly from inferior documentation and/or communication. It is particularly distressing when added to this there are injudicious, damaging comments from a colleague that fuel the flame.

To my mind the most grievous form of malpractice is the selling of one's services to plaintiffs' attorneys without regard to truth; this is gaining in frequency. Sadly, as competition increases, we have former colleagues who are making a career of such scurrilous activities. There are times, of course, even in the absence of actual malpractice, when in the interests of the patient and truth any one of us must testify against a colleague; there is frequently room for differences in interpretation and judgment, and all aspects of a case deserve airing. I am not speaking of that; I am speaking of professional witnesses whose souls are for sale. Unfortunately, the court seldom admits such information about witnesses, holding it to be irrelevant; professional witnesses are generally very persuasive.

Aside from scrupulous attention to individual professionalism, the only rational solution to the professional liability crisis is thorough-going tort reform; this is opposed by the trial lawyers, even though it received the support of the Bar. The state of Indiana, which has the lowest professional liability premiums in the country, does not allow claims to be brought against a provider of health care until it has been reviewed by a medical review panel, which is granted absolute immunity from civil liability. Among other provisions, there is a limitation on recovery, and there is a compensation fund administered by the Department of Insurance to pay claims in excess of individual coverage. Attorneys' fees are limited to 15% of recovery, and there is a statute of limitations of two years from the date of injury.

Because of opposition from the Trial Lawyers Association, a concerted effort by a large consortium, of which the TMA was only one member, failed in its attempts to have tort reform enacted by the last Tennessee legislature. Legislation will be reintroduced in the next legislature, and demands your best efforts in its behalf.

No reform, however, will ever protect anyone from lawsuits who fails in courtesy toward his patients and their families, who fails to communicate adequately with them, and who fails to document even what appear to be the most inconsequential details. Just bear in mind that nothing is inconsequential, no matter how seemingly trivial, if its failure will get you sued.

J.B.T.

The Dead Hand. . .

> Ah gits weary, an' sick o' trying;
> Ah'm tired o' livin', an' skeered o' dyin'—
> But ole man Ribber, he jes keeps rollin' along.
>
> —Jerome Kern
> *Showboat*

Whatever the negative aspects of medical practice, and these days they are often substantial, it is never boring. And a good thing, too.

Early in my life I had it ingrained in me that it was a good idea to brush my teeth every day. More bluntly, I had to. That was before Jack Benny made the Ipana toothpaste slogan a household tenet: See your dentist at least twice a year and use Ipana toothpaste twice a day. Once a day seemed like enough—but once was clearly necessary. Later, Pepsodent (or maybe it was earlier; I forget) got into the act with the smile of beauty and the smile of health; one of the first singing commercials went: "You wonder where the yellow went/When you brush your teeth with Pepsodent." You could say all that stuff was habit forming, but such habits, unlike some others—usually more pleasing, if more dangerous—are easy to break. Later on (much later—the young tend to take their smile of beauty and health for granted) I learned I needed to "floss" after brushing. It was not until I found my teeth took to hurting if I didn't floss, at least sometimes, that that habit took hold.

Did you ever stop to think how much of your time and energy you spend on your alimentary tract? Well, stop and think. One way or another, most of it—unless you live on a South Sea island where you can go out and pick your lunch free; few of you do. So, most of it. Those things add up. If sex (though not always called that) has been America's watchword (I don't of course imply that that is exclusively American), then "regularity" has been its password (pun possibly intended). Only the means of attaining it change. When I was growing up it was through calomel (which mercifully soon faded into oblivion) and—ugh!—castor oil, on to such milder things as Milk of Magnesia, Feen-a-mint, and Ex-Lax. Then there was Epsom Salts (a dose of) and Fleet's Phospho-soda (which is still around). I got most of those frequently. I swore that when I got away from home none of them would ever again pass my lips. Except to accommodate my friendly endoscopist, they mostly haven't. The lowly prune is sufficient unto the day, instead of the evil thereof. Now bran is in and laxatives are out. That's for the in between (the top and the bottom).

The Balm in Gilead to keep the bottom end happy is the psyllium seed, commonly known by its trade name Metamucil (sometimes derogated to metamucilage, which is what it tastes like). Its manufacturers (or more properly, packagers, since its manufacturer is the psyllium plant) have done a workman-like job of masking that with orange, lime, or what-not flavoring, and they have even done that without adding sugar. It is not the taste, then, that is objectionable.

It is the boredom. Before I have gotten my eyes open mornings I have washed my face, brushed my teeth, and shaved. I have been doing that so long it is as much a part of life as breathing. I have had to add, though, flossing, Metamucil, and bran mornings, and Metamucil, brushing, flossing, and exercising (another crash-ing bore) before I shower and go beddy-bye. Though that seems like a lot, it really isn't. Even so, I have a hard time being compliant. (Some of the "patients' rights" advocates are objecting to the use of the term "compliance," allowing that it suggests a master-slave relationship. Well, it is—but not slavery to the doctor, as they suggest. The compliance is, in my case, to the demands of an aging body, and in others to a sick one. It's a good term. When I fail to comply, who tells me first—my doctor? Not on your life: it isn't his drainpipes that get clogged.)

One morning recently I happened to overhear a part of one end of a telephone conversation between one of my associates and his 91-year-old mother. "Have you taken your heart medicine this morning? . . . Well, did you take it last night? . . . Well, have you taken your. . . ?" I have a friend who starts his day out by making up little packages of pills, and inscribing on each packet the time he is to take it; there are several pills in each one—more in some than in others. He is old, and he is not well (obviously), but he is functional. He spends a lot of his time keeping that way. "What do you do," I once asked him, "if you miss a dose?" "I throw it away," he said. He is a doctor.

I think I have found out why people die. I had my first brush last winter with Medicare. To begin with, despite all my efforts, I have never been able to coordinate my wife's and my payments for Part B, which doubles that trouble. The amount of paperwork generated by even a small hospitalization is incredible, and most of it I didn't understand, anyway; I doubt if anybody else did, either. And Medicare, since I am still working in a professional corporation, is not even my primary insurance. All I do with all that paperwork is give it to one of our employees who takes care of such things, and she takes care of those things. It is disquieting—the mildest term I can use for that particular terror—that once I retire, I will have to do all that myself. It is strong incentive to keep working.

That is not why people die, though—or not altogether. I think they just get tired of complying, and so they don't. (Obviously, some people die because one or another of their innards quits—I'm not talking about them.) They get tired of filling out forms they don't understand and sending them to ribbon clerks who don't understand them either. Their days become filled, as they do themselves, with pills and powders and (they think) pedants telling them to take their pills and powders. As the saying goes, it takes a heap o' livin' to make a house a home; once the playroom gets closed up, and the cupboard contains only powders and pills, comes time to evacuate. Few do it deliberately; a lot more just drift away.

The cost-benefit ratio, which is the "in" term these days, applies to other things than money. Some folks have more resilience than others—or perhaps just more curiosity—but when the excitement of living is exceeded by the boredom of compliance, or when, to use the vernacular, the pain in the ass is worse than the pain in the heart, unless they are more feared o' dyin' than they are tired o' livin', folks tend to give up.

The moral to this story (which will doubtless be lost on our younger colleagues) is not to bail out of your practice without a parachute; the life you save may be your own. There is one thing more deadly than the dead hand of bureaucracy. That is boredom—and that the practice of medicine isn't.

J.B.T.

A Golden Anniversary

> For those who turn to the high hills and follow wood trails . . . the date August 14, 1937 is of particular significance. On that day . . . a CCC [Civilian Conservation Corps] trail crew went through a last barrier of a dense scrub spruce and fir tangle out into broad, open trail. Beyond, north, white blazes and an open route led 1,868 miles to Katahdin [Maine]; similarly, south, 1,868 miles to Mt. Oglethorpe in Georgia. . . . Thus, some 16 years after its inception in 1921, the longest marked footpath in the world was initially opened. . . . Much still remains to be done. . . .
>
> —Myron H. Avery, 1938
> *In the Maine Woods*

There are always anniversaries of one sort or another, some better celebrated than others, even if not more significant. Both celebrated and significant this year is of course the 200th birthday of the United States Constitution, though whether what we have now is what was born 200 years ago is open to question. It has grown, to be sure—just as one would suspect of any child through experience, but as with the Emperor's new clothes, what you see is not necessarily what you get. What you and I get is what nine old lawyers see, and they through a glass darkly, as there is seldom consensus. But I digress.

I will also digress to remind you, or to tell you in case you never knew it, that this is the hundredth birthday of the flat phonograph record, though cylinders were used to reproduce sound before that. Unless you consider the compact disc (CD) the same thing only different, the flat phonograph record is about to go the way of the cylinder, being supplanted by audiotapes and CDs. Sound reproduction has come a long way, and is near picture-perfect—but not the record. So much for that.

Back in the Thirties, when we used to go to the small resort hamlet of Gatlinburg every spring to watch the rhododendron pop out, both the locals and the visitors, most of the latter city slickers from Knoxville who had summer homes there, and the rest of them city slickers from elsewhere (city slickers, like beauty, being only in the eye of the beholder) who were guests at one of the two hotels—the Mountain View Hotel and the Greystone Lodge—liked to sit around Ogle's store and listen to a bewhiskered old gentleman named Wiley Oakley talk about his exploits on the Appalachian Trail, the length of which (all 2,000 odd miles of it) he had walked two or three times. The time I heard him, somewhere around 1936, give or take a year or so, he was about to take off and do it again. Because of his long flowing white beard and my extreme youth, I thought Wiley Oakley must be nearly as old as God. It never occurred to me that except for God people that old don't hike the Appalachian Trail.

The Appalachian Trail is 50 years old this year—though the Appalachian trails it derives from are as old as human habitation in the region. As if loath to share its glory with another, nature refused to cooperate for the initial grand opening of the Trail in Maine, so that due to a snowstorm in Maine a later scheduled strip in Tennessee was finished first. She later refused cooperation again for a "reopening" ceremony following completion of the relocation in 1951 of a 118 mile strip in Virginia, where torrential rains flooded out the projected "all stops out" celebration. What started out on two occasions as a monumental fanfare wound up as mere whimpers that elicited only scant response from the media. The Appalachian Trail was finished. So what?

In fact, it was not finished, nor will it likely ever be. There are strips that proceed down Main Street that need to be relocated to a fitting permanent home. Land acquisitions therefore continue. Other strips are constantly being relocated for scenic purposes or for increased seclusion. The one mile strip of wilderness on either side of the Trail as projected by its planners is easy to accomplish where it winds through national parks and forests; in many other places it is an idealistic dream. More than 90% of the Trail is protected, though, thanks to the 1968 National Trails Systems Act, which designated the Appalachian Trail as the country's first National Scenic Trail. In 1978 Congress authorized $90 million for acquisition of corridor land at 125 acres per mile of trail, which would give 500 feet of protection on either side; it is a tremendously complex program requiring acquisition of more than 1,500 parcels of land in nine states.

The acquisition has progressed marvelously well, particularly considering that it has occurred during an era of political austerity toward public recreation projects. Since 1978, 621 miles have been protected through federal and state land acquisitions. Well, again, so what? Is it worth it?

If you have never been on the Trail, you owe it to yourself to try it out! Obviously, to complete the entire 2,100 miles requires dedication and stamina beyond what most of us can afford. I have hiked only a short segment, between Cade's Cove and Newfound Gap, and that more than once. There are, too, other trails equally fair and a lot easier. But "Appalachian Trail" has a sort of stentorian ring to it—as though it really *means* something. Maybe it's only snob appeal; *I've* been on the *Appalachian* Trail. Well, *I've.* Not much, to be sure, but I have. No telling how many have done the whole thing; 236 have since December, 1985.

Apparently the hoopla that fizzled in 1937 and 1951 has ignited along the Trail in 1987, as communities situated along the Trail commemorate its Golden Anniversary. It's something we don't hear much about "in the West" (when our Constitution was born, everything this side of "The Mountains" was the West).

If you have wondered who keeps up the Trail, well, it's the likes of you and me. It likely would never have gotten off the ground without the CCC, which was formed during the depression along semi-military lines to get ablebodied young men off the streets and set them to doing productive labor. With World War II, though, such governmental contributions ground to a halt, and since then, maintenance of the Trail has been carried on by members of Trail clubs that have grown up along the Trail. These dedicated men and women and boys and girls spend weekends and vacation time healing winter's wounds along the Trail, maintaining shelters, clearing under-brush, even picking up other people's trash, and so on. Those clubs are coordinated by the Appalachian Trail Conference, through which individuals can for $25 a year show their interest and contribute their part to the Trail—a pittance compared to the clubs' contributions. For it, you get an informative publication, *Appalachian Trailway News*, which carries information about the Trail, its environs, and nature and woods lore generally. You can also get maps and guidebooks.

And satisfaction; at least, I do. Write Appalachian Trail Conference, P.O. Box 807, Harpers Ferry, WV 25425, and Happy Trails to you!

J.B.T.

in memoriam

Fred Moore, Jr., age 67. Died August 17, 1987. Graduate of University of Tennessee College of Medicine. Member of Northwest Tennessee Academy of Medicine.

TMA Members Receive AMA Physician's Recognition Award

Thirteen TMA members qualified for the AMA Physician's Recognition Award during July 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Ronald B. Addlestone, M.D., Nashville
Daniel F. Beals, M.D., Knoxville
Patricia M. Bihl-Miranda, M.D., Nashville
John C. Brothers, M.D., Nashville
Donald R. Lovelace, M.D., Nashville
Harold A. McCormack, M.D., Memphis
Steven W. Neubauer, M.D., Nashville
Elsie P. Ollapally, M.D., Madison
David N. Orth, M.D., Nashville
Winston C. V. Parris, M.D., Nashville
Molly M. Peeler, M.D., Memphis
Robert L. Pettus, Jr., M.D., Madison
Clarence B. Watridge, M.D., Memphis

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

CONSOLIDATED MEDICAL ASSEMBLY OF WEST TENNESSEE
Marcus Troxell, M.D., Jackson
F. Earl Williamson, III, M.D., Jackson

KNOXVILLE ACADEMY OF MEDICINE
Ann S. Bevelhimer, M.D., Knoxville
Jeffrey S. Hecht, M.D., Knoxville
Thomas G. Higgins, M.D., Knoxville

MAURY COUNTY MEDICAL SOCIETY
Earl Quinton Parrott, M.D., Columbia
Hans P. Vogel, M.D., Columbia

MEMPHIS-SHELBY COUNTY MEDICAL SOCIETY
Thomas L. Abell, M.D., Memphis
Edward Atkinson, III, M.D., Memphis
Charles H. Brown, M.D., Memphis
Rommel Gabriel Childress, M.D., Memphis
Michael Steven Dragutsky, M.D., Memphis
Mahmoud Ali Eltorky, M.D., Memphis
Morris George, M.D., Memphis
Hollis H. Halford, III, M.D., Memphis
Stephen Hall Landy, M.D., Memphis
Moacir Schnapp, M.D., Memphis
Thomas B. Shelton, M.D., Memphis
Thomas Gregory Stovall, M.D., Memphis
Margaret Ryan Todd, M.D., Memphis

MONTGOMERY COUNTY MEDICAL SOCIETY
Karen Pitts Foster, M.D., Clarksville

NASHVILLE ACADEMY OF MEDICINE
Gordon Clay Akin, M.D., Nashville
Sarah Brown Aylor, M.D., Brentwood
Thomas Haile Callaway, M.D., Nashville
Sage Kinney Copeland, M.D., Nashville
Eva Agnes Dimitrov, M.D., Nashville
William M. Goodman, M.D., Madison
James Robert Hart, M.D., Brentwood
John Edward Keyser, III, M.D., Nashville
Albert Brant Lipscomb, M.D., Nashville
James P. Little, M.D., Cedar Hill
Ruth Barron Long, M.D., Nashville
Keith H. Loven, M.D., Nashville
Raymon S. Martin, III, M.D., Nashville
Robert Trigg McClellan, M.D., Nashville
Bonnie Mersky Miller, M.D., Nashville
Douglas Edward Norton, M.D., Nashville
William Charles Penley, M.D., Nashville
Charles Sidney Settle, M.D., Nashville
Paul W. Wheeler, M.D., Nashville
Cynthia C. Youree, M.D., Nashville

personal news

James H. Barker, M.D., Jackson, has been named a fellow of the American Academy of Facial Plastic and Reconstructive Surgeons.

James W. Davis, M.D., Chattanooga, has received the Kenneth L. Pickrell Award from the Southeastern Society of Plastic and Reconstructive Surgeons.

William F. Meacham, M.D., Nashville, has received the Cushing Medal, the highest award given by the American Association of Neurological Surgeons.

announcements

CALENDAR OF MEETINGS

NATIONAL

Nov. 1-4	American Physicians Art Association Meeting and Exhibit—San Antonio, Tex.
Nov. 1-4	Southern Medical Association—San Antonio, Tex.
Nov. 2-5	Interstate Postgraduate Medical Association—The Diplomat, Hollywood, Fla.
Nov. 2-7	American College of Emergency Physicians—San Francisco
Nov. 4-7	American Cancer Society—New York
Nov. 4-7	American Medical Writers Association—Drake Hotel, Chicago
Nov. 4-8	American Society of Cytology—New Orleans
Nov. 5-7	Society for the Scientific Study of Sex—Westin Peachtree Plaza, Atlanta
Nov. 6-7	American Association of Neurological Surgeons and Congress of Neurological Surgeons Joint Section on Trauma—Marriott Orlando World Center, Orlando, Fla.
Nov. 6-11	National Perinatal Association—Adam's Mark Hotel, St. Louis
Nov. 7-12	American Association of Blood Banks—Orlando, Fla.
Nov. 7-12	Association of American Medical Colleges—Washington Hilton, Washington, D.C.
Nov. 8-10	Association of American Physicians and Surgeons—New Orleans
Nov. 8-10	Association for Hospital Medical Education—Washington, D.C.
Nov. 8-12	American Academy of Ophthalmology—Dallas
Nov. 8-13	American Society of Maxillofacial Surgeons—Georgia World Congress Center, Atlanta
Nov. 8-13	American Society of Plastic and Reconstructive Surgeons—Atlanta
Nov. 8-13	Association of Military Surgeons of the United States—Las Vegas
Nov. 10-13	Association for Medical Education and Research in Substance Abuse—Crowne Plaza Hotel, Rockville, Md.
Nov. 11-15	Academy of Psychosomatic Medicine—Riviera Hotel, Las Vegas
Nov. 11-15	Christian Medical Foundation International—Tampa, Fla.
Nov. 11-15	International Congress of Gynecologic Endoscopy—Sheraton Palace, San Francisco
Nov. 12-14	Association of Clinical Scientists—Tampa, Fla.
Nov. 14	Association for the Advancement of Psychotherapy—New York
Nov. 14-18	American College of Allergists—Marriott Hotel/Copley Plaza, Boston
Nov. 16-18	American Society of Bariatric Physicians—Bally's Hotel, Las Vegas
Nov. 16-19	American Heart Association—Convention Center, Anaheim, Calif.
Nov. 18-22	Gerontological Society of America—Washington Hilton, Washington, D.C.
Nov. 21	Chinese-American Medical Society—New York
Nov. 28-Dec. 4	Radiological Society of North America—Palmer House, Chicago
Dec. 3-6	Eye-Bank Association of America—Boston
Dec. 3-6	Society for Ear, Nose and Throat Advances in Children—Fairmont Hotel, Denver
Dec. 6-9	American Epilepsy Society—Baltimore
Dec. 10-13	American Academy of Psychoanalysis—Scottsdale, Ariz.

STATE

Nov. 3-6	Tennessee Academy of Family Physicians 39th Annual Scientific Assembly—Gatlinburg
Nov. 13-15	Tennessee Psychiatric Association Fall Meeting (in conjunction with American Society for Adolescent Psychiatry)—Vanderbilt Plaza Hotel, Nashville.

TENNESSEE MEDICAL ASSOCIATION

153RD ANNUAL MEETING

April 13-16, 1988

Peabody Hotel, Memphis

Minutes of the Tennessee State Board of Medical Examiners Meetings

February 3-4, 1987

Members Present: I. Lee Arnold, M.D.
Duane C. Budd, M.D., Acting Chairman
William W. Cloud, M.D.
Paul A. Johnson, M.D.
Members Absent: Phillip W. Hayes, M.D.

The meeting was called to order at 8:35 AM by Dr. Duane C. Budd, acting chairman.

The first item of business was election of officers. The following were unanimously elected: Drs. Duane C. Budd, president; William W. Cloud, secretary; Paul A. Johnson, consultant.

Reciprocity applications for Drs. **Vinary Vermani, Susan E. Besser, Truman L. Chastain, Edward P. Scott,** and **Howard F. Webb, III** were unanimously approved. The reciprocity application of Dr. **George M. Nowak** was approved with the stipulation that he would continue under contract with TMA's Impaired Physician Program. The reciprocity application for Dr. **Nicholas N. Dzebolo** was deferred until proof is shown that the investigation in Mississippi has been completed and his license there is cleared. The reciprocity applications for Drs. **Cortez E. McFarland, David J. Neilsen, Ajay Sobti,** and **Jerry B. Williams** were tabled since they were not present.

There was a discussion concerning the annual meeting of the Federation of State Medical Boards in April. The Board voted to request out-of-state travel approval for Dr. Budd, Dr. Cloud and Mrs. Blair. This is an important meeting and very beneficial to the Board and the administrative staff to meet with representatives of the other states and to share problems and solutions.

X-ray Operators

Recommendations from Mr. Michael D. Huggins and Dr. Fredia Wadley, who were representing the Commissioner's office, follow: (1) that the original task force be reconstituted, with a radiology consultant to the Board; (2) that the task force be involved with curriculum and examination of the two new categories—abdomen and spine, and skull and sinuses; and (3) that a practicum be given at end of training course as a part of the examination. The Board directed the administrative staff to go ahead with certification for the ones who passed the examination and to consider the ones who had 10 years' experience out of the last 15 to have met the requirements of the grandfather clause.

Hearings

The hearings for Drs. **Harvey Anderson, C. M. Baylor,** and **D. P. Shupe** were continued.

Dr. **Harry Helm** was charged with unprofessional, dishonorable, or unethical conduct; gross malpractice, or a pattern of continued or repeated malpractice, ignorance, negligence or incompetence in the course of medical practice; dispensing, prescribing, or otherwise distributing any controlled substance or any drug not in the course of professional practice, or not in good faith to relieve pain and suffering, or not to cure an ailment, physical infirmity or disease; dispensing, prescribing or otherwise distributing to any person a controlled substance or other drug if such person is addicted to the habit of using said controlled substances without making a bona fide effort to cure the habit of such patient and dispensing, prescribing or otherwise distributing any controlled substance or any other drug to any person in violation of any law of the State or of the United States of America. It being the duty and responsibility of the Board of Medical Examiners to regulate the practice of medicine in such manner as to promote and protect the public health, safety and welfare, the Board indicated its findings and conclusions, following which, upon application of respondent, in consideration of the Board taking no other action at this time, it was ordered, by agreement, that the respondent shall retire his license pursuant to TCA 63-1-111 and forever retire from the practice of medicine. The respondent has further agreed, after discussion with his attorney, that he will not ever apply for reinstatement of his license or to practice medicine in the future.

Dr. **Francis J. Pimentle** was charged with unprofessional, dishonorable, or unethical conduct; violation or attempted violation, directly or indirectly, or assisting in or abetting the violation of, or conspiring to violate, any provision of TCA 63-6-101 et seq, or any lawful order of the Board issued pursuant thereto, or any criminal statute of the State of Tennessee; gross malpractice or a pattern of continued or repeated malpractice, ignorance, negligence or incompetence in the course of medical practice; dispensing, prescribing or otherwise distributing any controlled substance or any other drug not in the course of professional practice, or not in good faith to relieve pain and suffering, or not to cure an ailment, physical infirmity or disease; dispensing, prescribing or otherwise distributing to any person a controlled substance or other drug if such person is addicted to the habit of using said controlled substances without making a bona fide effort to cure the habit of such patient; and dispensing, prescribing, or otherwise distributing any controlled substance or any other drug to any person in violation of any law of the State or of the United States of America. Based on findings of fact, the Board ordered that the respondent be placed on probation for a period of one year from the effective date of the order. The conditions of this probation are as follows: (1) Respondent shall submit a copy of each prescription he issues to the consultant for the Board of Medical Examiners for the following year. Said prescriptions shall be submitted on a quarterly basis; (2) Respondent shall remove all prescription pads and blanks from his residence and secure the same in an alternate location under lock and key. He shall further take whatever additional precautions that may be necessary to prevent his wife from gaining access to the same; and (3) Respondent shall secure drug and psychiatric counseling for his wife. She will be required to participate in the same for a period of one year following the effective date of this order.

Dr. **Joseph P. Moss** entered into an agreed order on July 8, 1986, when the Board found him guilty of personal abuse and misuse of controlled substances to such an extent and in such a manner that his condition had required medical intervention on more than one occasion. The agreed order was as follows: (1) The license to practice medicine previously issued to Joe P. Moss, M.D., is placed on probation for two years; (2) Dr. Moss shall submit to random drug screens as requested by his treating physician and the TMA Impaired Physician Program; (3) Dr. Moss shall sign and enter into a contract with the Impaired Physician Program for the duration of the probation heretofore imposed; and (4) If Dr. Moss fails to comply with the terms of his contract with the Impaired Physician Program or loses the advocacy of that Program, he agrees that the Program shall report such to the Board and such violation of the terms of the contract or the loss of advocacy shall be considered a violation of the provisions of this order. The charges now are that Dr. Moss violated the agreed order on several occasions in 1986 by failing to comply with the terms of his contract with the Impaired Physician Program, that he has personally misused controlled substances since July 8, 1986 and lost the advocacy of the Impaired Physician Program. The Board found him guilty and voted unanimously to suspend his license to practice medicine indefinitely. This was a default judgment since Dr. Moss is now in treatment in Mississippi and was not present.

Dr. **Bobby Watts** was charged with violation or attempted violation, directly or indirectly, or assisting in or abetting the violation of, or conspiring to violate, any provision of TCA 63-6-101 et seq, or any lawful order of the Board issued pursuant thereto or any criminal statute of the State of Tennessee; making false statements or representations, being guilty of fraud or deceit in the practice of medicine; gross malpractice, or a pattern of continued or repeated malpractice, ignorance, negligence or incompetence in the course of medical practice; dispensing, prescribing, or otherwise distributing any controlled substance or any other drug not in the course of professional practice, or not in good faith to relieve pain and suffering, or not to cure an ailment, physical infirmity or disease; dispensing, prescribing or otherwise distributing to any person a controlled substance or other drug if such person is addicted to the habit of using said controlled substances without making a bona fide effort to cure the habit of such patient; dispensing, prescribing, or otherwise distributing any controlled substance or any other drug to any person in violation of any law of the State or of the United States of America; unprofessional, dishonorable, or unethical conduct; the advertising of medical business in which untrue or misleading statements are made, or causing the publication or circulation of fraudulent advertising relative to any disease, human ailment, or conditions; and any person who treats or professes to treat another is regarded as practicing medicine. If said person does so without first complying with the provisions of the Medical Practice Act, the provisions of the Physician Assistant Act, or the Nursing Practice Act is guilty of violating the provisions of TCA 63-6-203 and 63-6-204. The hearing was not completed and will be continued at the next Board meeting.

A Board meeting to discuss x-ray operators rules was set for March 21, 1987. April 14 and 15, 1987, were set as the next meeting dates for other business and to continue the Bobby Watts hearing.

There being no further business, the meeting adjourned at 5:00 PM.

March 21, 1987

Members Present: Duane C. Budd, M.D., President
William W. Cloud, M.D., Secretary
Paul A. Johnson, M.D., Consultant
I. Lee Arnold, M.D.
Members Absent: Phillip W. Hayes, M.D.

Dr. Duane Budd, president, called the meeting to order at 9:00 AM.

Dr. Budd gave background information on the x-ray operator regulations for the benefit of the new Board members and the others present.

X-ray Operators

There was testimony given by the people present that was both for and against the non-R.T. x-ray operator in a physician's office and the rules the Board was trying to promulgate.

Dr. Budd brought out that the Board was charged with the responsibility of protecting the public and certainly did not wish to make any rules that would endanger the public. The Board asked for figures as to the danger of too much radiation given in a physician's office versus the radiation the patient would receive in a hospital or other setting and determined no statistics were given. The testimony from the practicing physicians in the state is that they have been giving x-rays in their offices for years without known hazards to their patients, with benefits far outweighing any hazard that has been suggested in terms of finances; if you go to the hospital it costs three times as much as it does in the offices. The Board has asked for figures showing increased risks in the physician's office by non-R.T. personnel operating x-ray machines, and so far no one came forward with these figures.

After listening to all the testimony, the Board reached the following decisions:

(1) To leave the grandfather clause as it appears in the Rules on file—with 10 years' experience out of the last 15 years.

(2) Reconstitute the Task Force to include the four originally on the Task Force plus one each from the following specialties: family practice, ENT, urology and internal medicine. The otolaryngologist would be Dr. Richard W. Babin and the urologist would be Dr. W. B. Crenshaw. The Tennessee Society of Internal Medicine and the Tennessee Association of Family Practitioners would be requested to submit three names each from which the Board could appoint the family practitioner and the specialist in internal medicine.

The purpose of reconstituting the Task Force was to study the proposed changes in the rules which added the certification in abdomen and spine and skull and sinuses. The Task Force would then make recommendations to the Board concerning these two new categories and any other recommendations they felt were warranted concerning the rules already in place which address chest and extremities. These recommendations would include curriculum and number of hours of instruction and supervised clinical practice.

(3) The Task Force would also give three names of radiologists as recommendations to the Board in order for the Board to appoint one person from the list to serve as a consultant to the Board.

(4) The consultant's duties would be to advise the Board in radiology matters and, twice a year, construct an examination from the pool of questions submitted by the course directors and from his own knowledge and pool of information. Dr. Budd suggested that the examination could consist of: (a) 15 questions on basic radiation and biology, (b) 10 questions on chest anatomy and positioning, (c) 10 questions on extremities anatomy and positioning, (d) 10 questions on skull and sinuses anatomy and positioning, and (e) 10 questions on abdomen positioning and anatomy.

(5) The Board has had numerous complaints concerning Mr. Archie Gore's course which the Board previously approved and is considering withdrawing approval. Some of the complaints were that the course was shallow and superficial, outdated materials were used, instruction was poor, etc. There is also information that Mr. Gore is offering courses in skull and sinuses and spine and abdomen, even though he had been told these had not been approved. There is a possibility that the doctors think the rules are in place and are sending their x-ray operators to these courses. The decision of the Board was to hire an R.T. to assist in investigating the course and to ask Sylvia Moore of the Nursing Board to assist the R.T. in the assessment of the course since Ms. Moore approves curriculum for nursing programs. Texas should be contacted since the course has also been taught there and in other states. When the investigation is completed, a report will be made to the Board, perhaps by May or June.

(6) The Board voted to terminate the contract with A.R.R.T. for the Limited Scope examination at the end of the present contract, June 30, 1987.

(7) The Board voted to add continuing education requirements and the monitoring process to the considerations by the Task Force.

(8) A letter will be drafted from the Board of Medical Examiners to Radiological Health to request that when their inspectors go to the doctor's office to inspect the x-ray equipment, they would also check the operators as to certification requirements and technical competence as set out in the rules of the Board. In addition, the Board wants a copy of the report that is sent to the doctor concerning the monitoring of x-ray exposure for the operators (wearing of monitoring badges).

(9) The Board also voted to require either a high school diploma or GED as documentation of being a high school graduate and that this would be required before anyone could be certified.

Applications

The Board approved the reciprocity application, pending updated information which includes new state clearances, for Dr. **James Naifeh,** which had been previously denied because of his training. In the opinion of the Board, Dr. Naifeh now meets the Board's requirements since he has received two years of additional training which was equivalent to two years in an approved residency program.

There being no further business, the meeting was adjourned at 3:00 PM.

MOVING? Send Us Your Address

Please notify us six weeks in advance.

Old Address

Name______________________________

Address______________________________

City/State/Zip______________________________

New Address

Address______________________________

City/State/Zip______________________________

Effective Date of New Address____________________

Send to: TMA, 112 Louise Ave., Nashville, TN 37203

TMA Membership Information and Statistics—1987

CAROLYN SANDLIN
Membership Director

Dues Information

Bills for the 1988 local, state, and American Medical Association dues will be mailed to the membership this month. Tennessee Medical Association dues remain the same as current 1987 dues ($220) with $25 allotted to the Student Education Fund and $15 allotted to the Impaired Physician Program.

1988 Dues to the Tennessee Medical Association

Active	$220
Active (1st and 2nd year in practice)	110
Resident	40
Retired (working less than 20 hours per week)	110

TMA dues are reduced by one-half for physicians new to the Association joining at mid-year (after July 1)

Dues Exempt Membership Categories for TMA—1988:

Veteran members (over age 70); *Retired* members (age 65-70—working 0 hours per week); *Disabled* members (those physicians who are unable to practice medicine); *Associate* members (those physicians who are serving in the armed forces or the public health service); *Honorary* members (special membership voted by the Board of Trustees); and *Student* members (limited to Tennessee medical schools).

Physicians who were exempt from dues in 1987 because of retirement (over age 65, working less than 20 hours per week) will retain their dues exempt status under the grandfather clause.

1988 Dues to the American Medical Association

Active	$375
Active (1st year in practice)	187
Active (2nd year in practice)	281
Resident	45
Retired (working less than 20 hours per week)	75
Over age 70 (working 1-20 hours per week)	187
Military	250
Student	20

AMA dues are reduced by one-half for physicians joining the Association at mid-year (after July 1)

Dues Exempt Membership Categories for AMA—1988:

Veteran members (those members over age 70 and fully retired—working 0 hours per week); *Disabled* members (those physicians who are unable to practice medicine or are experiencing financial hardship).

All physicians who were previously certified as exempt from dues under former AMA categories will remain exempt under the grandfather clause.

All membership privileges are extended the dues exempt membership by both the TMA and AMA, with the exception of the AMA publications. Dues exempt members are offered a reduced rate for these publications. Current year membership cards are sent by both TMA and AMA to the dues exempt members.

FIGURE 1

NUMBER OF TMA MEMBERS IN LISTED SPECIALTIES*

Internal Medicine	784
Family Practice	569
General Surgery	505
Obstetrics/Gynecology	363
Pediatrics	308
Anesthesiology	276
General Practice	274
Orthopedic Surgery	271
Radiology	228
Ophthalmology	223
Psychiatry	191
Pathology	191
Urology	162
Otorhinolaryngology	130
Cardiovascular Diseases	104
Diagnostic Radiology	104
Emergency Medicine	90
Neurological Surgery	89
Dermatology	78
Plastic Surgery	72
Thoracic Surgery	59
Neurology	58
Gastroenterology	52
Pulmonary Diseases	43
Gynecology	42
Oncology	35
Cardiovascular Surgery	31
Occupational Medicine	29
Public Health	28
Nephrology	20
Child Psychiatry	19
Rheumatology	19
Hematology	17
Therapeutic Radiology	15
Allergy/Immunology	13
Pediatric Surgery	13
General Preventive Med	11
Colon & Rectal Surgery	11
Otology	9
Nuclear Medicine	8
Physical Med & Rehab	8
Pediatric Allergy	7
Endocrinology	6
Pediatric Cardiology	5
Child Neurology	5
Neonatal-Perinatal Med	5
Diabetes	4
Aerospace Medicine	4
Obstetrics	3
Geriatrics	3
Other	41

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*Only primary specialties listed in this study.

All TMA members must be members of a component medical society. Each society certifies its members to the TMA. The TMA currently bills for local, state, and AMA dues on behalf of 45 component societies across the state; the remaining six societies bill their membership for all dues, reporting TMA and AMA dues to the TMA office. The TMA reports all AMA dues received to the Chicago office of the AMA each month.

Statistics

The TMA has 4,975 dues paying members and 936 physicians who are exempt from dues (figures as of July 13, 1987). Over one-half (56%) of the TMA members are board certified in their specialty. *Figure 1* gives the number of physicians in listed specialties as indicated by the Licensing Board for the Healing Arts for the State of Tennessee or by the physician.

Figure 2 provides a study in age distribution. Very little change is noted from 1986; the over age 65 group has increased by only 0.8% and the 35 to 44 age group continues to comprise 30% of the TMA membership. Female physicans number 339, up from 323 last year, making up 5% of the TMA membership.

Our statistics on the medical schools attended by the TMA membership remain the same as in 1986 (56%). Currently, 2,614 members have (or will) graduated from the University of Tennessee, Memphis College of Medicine, 626 from Vanderbilt University School of Medicine, 58 from Meharry Medical College, and 7 from Quillen-Dishner East Tennessee State University College of Medicine. *Figure 3* lists all of the medical schools in the United States and Canada represented in the TMA membership.

FIGURE 2

TMA MEMBERSHIP PROFILE BY AGE

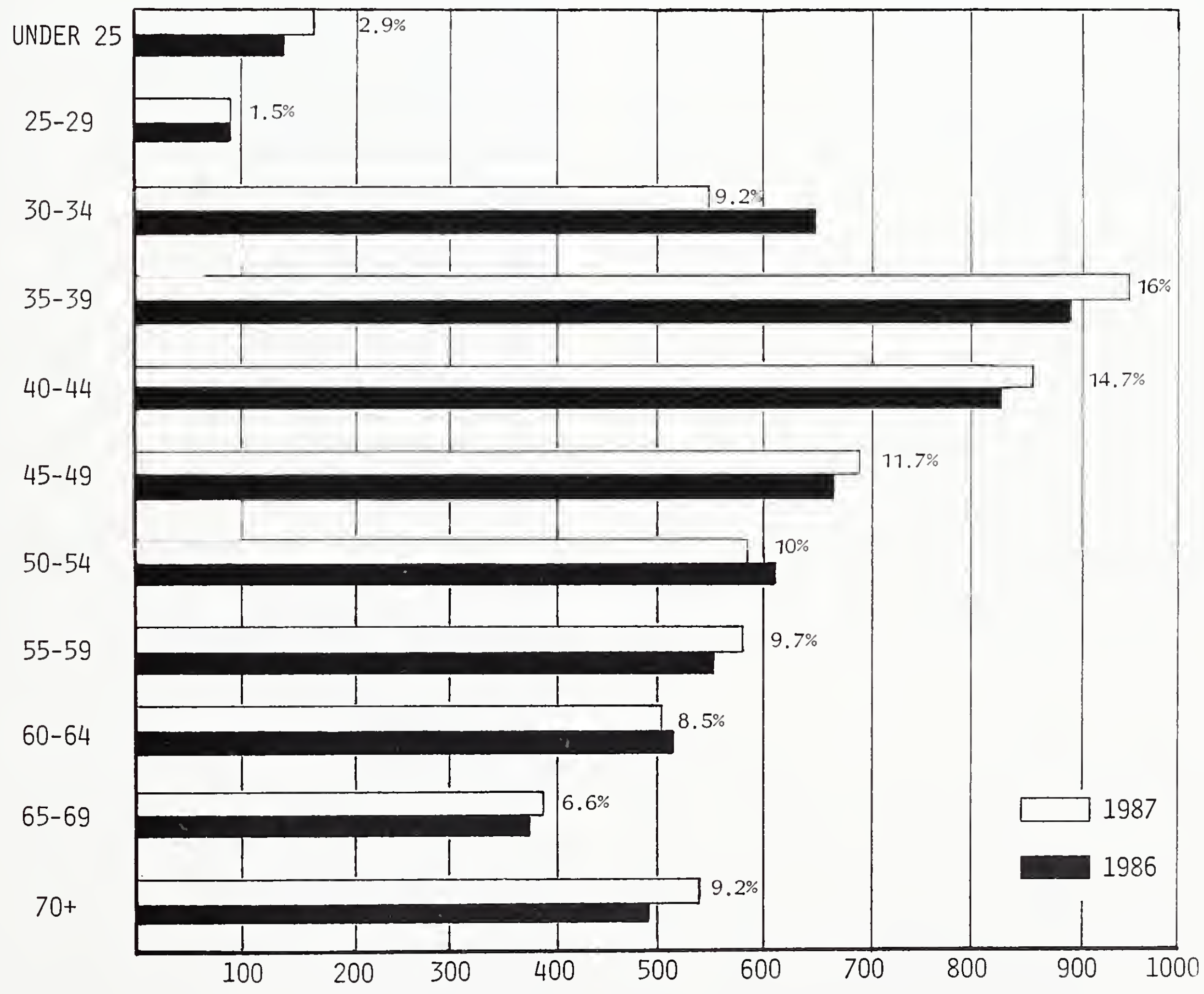

FIGURE 3

MEDICAL SCHOOLS IN UNITED STATES AND CANADA REPRESENTED IN TMA MEMBERSHIP

University of Tennessee/TN	2,614
Vanderbilt University/TN	626
Tulane University/LA	99
Loma Linda University/CA	88
University of Louisville/KY	83
Emory University/GA	80
University of Virginia/VA	76
University of Arkansas/AR	73
University of Mississippi/MS	72
Duke University/NC	70
Medical College of Georgia/GA	65
University of Alabama/AL	62
Meharry Medical College/TN	58
Johns Hopkins University/MD	57
Medical College of Virginia/VA	56
Harvard Medical School/MA	46
Indiana University/IN	44
University of Pennsylvania/PA	42
Washington University/MO	42
Bowman Gray School of Medicine/NC	41
Medical College of South Carolina/SC	41
Louisiana State University, New Orleans/LA	40
University of North Carolina/NC	38
Ohio State University/OH	32
University of Illinois/IL	32
Baylor College of Medicine/TX	32
University of Michigan/MI	27
University of Texas, Galveston/TX	27
George Washington University/DC	27
Northwestern University/IL	26
Jefferson Medical College/PA	26
University of Kentucky/KY	26
University of Kansas/KS	25
Columbia University/NY	22
University of Oklahoma/OK	22
University of Cincinnati/OH	21
St. Louis University/MO	21
Cornell University/NY	21
Wayne State University/MI	21
Temple University/PA	19
University of Texas, Southwestern/TX	19
State University of New York, Brooklyn/NY	18
University of Pittsburgh/PA	18
Case Western Reserve University/OH	17
Georgetown University/DC	16
West Virginia University/WV	16
University of Wisconsin/WI	16
Medical College of Wisconsin/WI	16
University of Florida/FL	15
University of Rochester/NY	15
University of Miami/FL	15
Pritzker School of Medicine/IL	14
New York University/NY	13
University of Maryland/MD	13
University of Iowa/IA	13
Tufts University/MA	13
Hahnemann Medical College/PA	13
University of Missouri, Columbia/MO	13
University of Nebraska/NE	12
McGill University/Canada	12
University of Minnesota/MN	12
Yale University/CT	11
New York Medical College/NY	10
University of Colorado/CO	10
Loyola University/IL	10
Medical College of Pennsylvania/PA	9
Howard University/DC	8
Boston University/MA	8
Albany Medical College/NY	8
University of South Florida/FL	8
State University of New York, Buffalo/NY	7
University of Vermont/VT	7
University of Ottawa/Canada	7
East Tennessee State University/TN	7
Dalhousie University/Canada	6
University of Washington/WA	6
Creighton University/NE	6
State University of New York, Syracuse/NY	6
Mount Sinai School of Medicine/NY	6
Rush Medical College/IL	6
University of California, San Francisco/CA	5
University of South Alabama/AL	5
University of Western Ontario/Canada	5
Mayo Medical School/MN	5
University of Southern California/CA	5
University of Texas, San Antonio/TX	4
Chicago Medical College/IL	4
University of Alberta/Canada	4
Pennsylvania State University/PA	4
Southern Illinois Medical School/IL	4
Michigan State University/MI	4
University of Toronto/Canada	4
Texas Tech University/TX	4
University of California, Los Angeles/CA	3
University of Oregon/OR	3
New Jersey Medical School/NJ	3
University of Texas, Houston/TX	3
Louisiana State University, Shreveport/LA	3
University of Manitoba/Canada	3
University of Connecticut/CT	3
Medical College of Ohio, Toledo/OH	2
University of New Mexico/NM	2
University of Utah/UT	2
Memorial University of Newfoundland/Canada	2
University of California, Irvine/CA	2
University of Puerto Rico/PR	2
Albert Einstein College of Medicine/NY	2
Stanford University/CA	1
McMaster University/Canada	1
East Virginia Medical School/VA	1
University of Arizona/AZ	1
University of Calgary/Canada	1
Laval University/Canada	1
University of Montreal/Canada	1
University of North Dakota/ND	1
Uniformed Services University, Bethesda/MD	1
Dartmouth Medical School/NH	1
Wright University/OH	1
Texas A&M University/TX	1
Foreign Medical Schools	412

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Hyperparathyroidism at a Tertiary Care Facility

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Introduction

In recent years, with the advent of routine serum calcium screening, an apparent increased incidence of hyperparathyroidism has occurred, and with this the discovery of a large "asymptomatic" subcategory. There is disagreement concerning the significance and therapy of an asymptomatic patient whose hypercalcemia and elevated parathyroid hormone levels are discovered incidentally.[1,2] In this study we present the results of a retrospective study of patients diagnosed as having hyperparathyroidism. In order to assess the "changing face" of the disease, we were particularly interested in the clinical presentation of our patients.

Materials and Methods

A retrospective review of patients diagnosed and treated for hyperparathyroidism was undertaken at Baroness Erlanger Hospital, a 780-bed tertiary care facility located in Chattanooga. Medical information on all patients with a diagnosis of hyperparathyroidism from July 1, 1980 to Dec. 31, 1985 was retrieved from the patients' medical records, and then collated and evaluated.

Laboratory values, most notably serum calcium, phosphate, chloride, and alkaline phosphatase, were all recorded from studies performed during the hospitalizations under study. The sole exception to use of current laboratory results was the parathyroid hormone immunoassay (iPTH), which was often performed prior to admission. The iPTH assay was performed at various outside laboratories utilizing different assay methods with different values for normal results.

In this study, by utilizing the term "metabolic bone disease," we intend to indicate only that the patient had some degree of demineralization secondary to his disease process without intending to indicate its severity. Its use is necessary because of the large number of radiologists involved in reading the films, and also because of the great diversity of films ordered (skull, hand, vertebrae, and others) to assess bony involvement.

Patients were divided into three study groups: group I includes those who underwent surgery for primary hyperparathyroidism; group II includes those who underwent surgery for hyperparathyroidism due to chronic renal failure; group III includes those with hyperparathyroidism who received no surgery. Standard described techniques of parathyroidectomy were utilized in all group I cases.[3] Parathyroidectomy with autotransplantation to the forearm was performed in all but one

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of the group II patients; one group II patient had a subtotal parathyroidectomy.[4]

Results

The age, sex, and presenting signs and symptoms are listed in Tables 1 and 2 respectively.

Clinical laboratory data at diagnosis and on postoperative day 4 are presented in Table 3. For group I, 91.3% of serum calcium levels at diagnosis were greater than 10.5 mg/dl. Postoperatively, serum calcium levels dropped for all group I patients. Serum chloride was not measured postoperatively often enough in group I patients to discern significant change.

Patients with metabolic bone disease are listed in Table 2. All of these patients had an elevated serum alkaline phosphatase. An iPTH assay was completed in 86.9% of group I patients, 75.0% of group II patients, and 91.7% of group III patients, and was elevated in all cases measured.

At surgery, of the 23 group I patients, 18 had a single adenoma, four had multiple adenomas or hyperplasia, and one had no evidence of glandular pathology. In 44.4%, the location of single adenomas was in the right superior pole, in 22.2% in the right inferior pole, in 27.8% in the left inferior pole, and in 4.3% in the left superior pole. Complications for both group I and group II patients are listed in Table 4.

One group II patient remained significantly hypocalcemic until he died of unrelated causes six weeks postoperatively. Another group II patient had an acute abdomen postoperatively; at exploratory laparotomy it proved to be a ruptured ovarian cyst. A third group II patient suffered from hematoma formation and bleeding, presumed to be due to chronic renal failure and resultant bleeding diathesis. Group II patients were treated with high calcium dialysates along with oral and intravenous calcium until their autografts began to function. Patients listed in Table 4 who experienced serious hypocalcemia manifested by tetany and seizures were administered 1,25 dihydrocholecalciferol.

During the period of this study, 136,347 patients were admitted to Baroness Erlanger Hospital, which yields an incidence of 26 cases of primary hyperparathyroidism per 100,000.

Discussion

Hyperparathyroidism has often been referred to as "stone and bone" disease, but this study supports the findings of others that the clinical presentation of hyperparathyroidism has a broader spectrum.[5,6] Our study reaffirms that a majority of primary hyperparathyroid patients are postmenopausal women. While a significant number of patients suffered from calculi or metabolic bone disease, our study also revealed an important number of patients presenting with hypertension, muscle weakness and fatigue, and mental changes. As expected, a significant number were asymptomatic (Table 2).

The high percentage of hyperparathyroid patients in this study with hypertension could well be coincidental. Although hyperparathyroidism has occasionally been suggested as an etiology of hypertension, no direct relationship has ever been proven, and currently the association of hypertension and hyperparathyroidism is considered circumstantial by some authors.[5]

Muscle weakness and fatigue have long been presenting symptoms of hyperparathyroidism, but until recently this relationship was recognized by only a minority of physicians. As primary hyperparathyroidism is diagnosed earlier in the disease

TABLE 1

AGE AND SEX CHARACTERISTICS OF THE STUDY GROUPS

	Group I	Group II	Group III
Male	7 (30.4%)	5 (27.8%)	2 (16.7%)
Age	61.5 ± 14.2	39.0 ± 10.6	74.0 ± 4.0
Female	16 (69.6%)	13 (72.3%)	10 (83.3%)
Age	58.9 ± 14.2	41.0 ± 16.0	67.8 ± 12.4

TABLE 2

CLINICAL PRESENTATION OF THE STUDY GROUPS*

	Group I	Group II	Group III
Hypertension	73.9	37.5	50.0
Pruritus	0	16.6	0
Calculi	30.4	6.25	41.67
Polyuria/Nocturia	26.0	—	8.3
Muscle weakness	30.4	18.75	8.3
Fatigue	26.0	6.25	0
Mental status changes	26.0	0	25.0
Peptic ulcer disease	17.4	6.25	8.33
Nausea and vomiting	13.0	0	8.33
Constipation	8.7	6.25	16.67
Bone pain	13.0	68.75	16.67
Metabolic bone disease	34.78	87.5	8.3
Asymptomatic	30.43	0	41.67

*Numbers represent percentage of patients within study groups with the presenting sign or symptom.

TABLE 3

LABORATORY DATA OF STUDY GROUPS*

	Group I	Group II	Group III
Calcium, at diagnosis	11.66 ± 0.96	9.34 ± 0.81	10.99 ± 1.26
Calcium, postoperative day 4	8.37 ± 0.49	8.07 ± 1.32	—
Calcium, % change	26.37% decrease	13.26% decrease	—
Phosphate, at diagnosis	2.78 ± 0.46	5.92 ± 2.45	3.08 ± 0.83
Phosphate, postoperative day 4	3.81 ± 0.80	3.56 ± 1.57	—
Phosphate, % change	41.75% increase	36.90% decrease	—
Chloride, at diagnosis	106.89 ± 3.01	97.72 ± 2.68	104.58 ± 3.58
Cl/P ratio > 33	83%	6.2%	50%
iPTH			
measured	19/23	14/18	11/12
elevated	100%	100%	100%

*Normal Values: serum calcium 8.5 to 10.5 mg/dl
serum phosphate 2.5 to 4.5 mg/dl
serum chloride 95 to 105 mEq/L

process by routine serum calcium screening, physicians must become cognizant of the more subtle effects of the hyperparathyroid state.

Mental disturbances have been increasingly associated with hyperparathyroidism,[5,7] and in our study, 25% of group I and 26% of group II had mental status changes noted on the admission history and physical. Since no standardized psychological profile was performed, the incidence could well be higher. Although not enough patients were examined postoperatively to arrive at any statistically significant evaluation of the effect of the operation on the patient's mental status changes, postoperative notes indicated that several patients experienced profound recovery of "lost" mental functions. No group II patients were reported to have changes in mental status preoperatively, likely due to an incomplete history and physical, although there may also be an unknown physiologic explanation.

Patients with hypercalcemia and hyperparathormonemia but without obvious effects of their disease process have been receiving greater attention, as this group has been diagnosed with increasing frequency with the advent of routine serum calcium screening. In our study, 30.4% of group I patients and 41.7% of group III patients were asymptomatic.

Our incidence of 34.78% of group I patients having some degree of demineralization associated with metabolic bone disease is much higher than has recently been reported, and is actually more consistent with reports published prior to the advent of routine serum calcium screening describing the incidence of metabolic bone disease. Also our incidence of 26 cases of primary hyperparathyroidism per 100,000 is much lower than has recently been reported (typically 100 to 300 per 100,000). This incidence is also similar to reports prior to the advent of routine serum calcium screening.[5] One must therefore question whether primary hyperparathyroidism, as evaluated by this study, has been diagnosed as early as possible in our geographic area.

Primary hyperparathyroidism has been reported previously to cause hypercalcemia, low to low normal serum phosphate, and high to high normal serum chloride[8]; our study supports this finding. One attempt to relate these variables has been the Cl/P ratio, which has been suggested as a mechanism by which one can differentiate primary hyperparathyroidism from other causes of hypercalcemia. Although initial reports indicated that a Cl/P ratio greater than 33 could detect 96% of hyperparathyroid patients and correctly differentiate them from other causes of hypercalcemia,[9] later reports did not find the Cl/P ratio to be this sensitive.[10,11] Our experience is more in

TABLE 4

COMPLICATIONS OF HOSPITALIZATION

	Group I	Group II
Hypocalcemia	7/23	18/18
Serious hypocalcemia*	1/7	3/18 (1 permanent)
Other Complications		
Digoxin toxicity	1/23	—
Postoperative bleeding, requiring reoperation and emergency readmission	—	1/18
Ruptured ovarian cyst	—	1/18

*As defined in text.

agreement with the latter, in that 83% of group I patients had a Cl/P ratio greater than 33. This result indicates that some confidence can be attributed to this ratio but it is not infallible.

Our group II patients showed findings consistent with previously published reports of secondary hyperparathyroidism, with elevated serum phosphate leading to diminished serum calcium and a high parathormone level.[8] As expected, chloride levels were low normal. Autotransplantation of parathyroid slices to the forearm performed well, and supplemental calcium was necessary in most cases only in the immediate postoperative period.

For a variety of reasons, patients in group III received no surgery. Some patients were considered poor surgical risks (most often due to poor cardiovascular or pulmonary status). Others simply refused surgery, and surgery was not offered to some who were asymptomatic. There was great diversity in the clinical presentation of this group. All had hyperparathyroidism established as a result of hypercalcemia, with concomitant increase in iPTH levels.

Acknowledgment:

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HELP FOR IMPAIRED PHYSICIANS

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Adolescent Suicide in East Tennessee

A Birth Cohort Study

WILLIAM L. MADEN, M.D.

The steadily increasing national suicide rate has been documented by Frederick[1] in his review article and by Murphy and Wetzel[2] in their study of suicide risk of birth cohorts.

Suicide has been identified in the past as an urban problem and a problem of the West and North.[3-5] *The goal* of this study is to determine the extent to which the suicide rate in East Tennessee corresponds to national trends. *The method* used in this study is that of birth cohort analysis. This method produces more precise information regarding trends and is readily com-

TABLE 1

SUICIDE RATES FOR WHITE MEN IN EAST TENNESSEE AND THE UNITED STATES

In East Tennessee

Year of Entry Into Study	Mean (SD) Annual Suicide Rates for Age Groups of Individual Cohorts[*] 15-19 yr	20-24 yr	25-29 yr	30-34 yr	35-39 yr	40-44 yr	45-49 yr	ANOVA Within Each Cohort as it Aged[†] Corrected χ^2	P
1949-1953	2.1	8.5	11.8	10.4	19.5	21.5	19.5	14.1	<.05
1954-1958	2.9	17.3	9.2	17.5	35.8	31.3	—	23.1	<.001
1959-1963	4.2	9.5	14.9	21.9	26.2	—		16.2	<.01
1964-1968	5.2	21.4	27.0	20.8	—			11.2	<.05
1969-1973	9.6	29.8	25.6	—				9.6	<.01
1974-1978	12.1	24.3	—					7.0	<.01
1979-1983	12.2	—							
ANOVA between cohorts at specific ages[†]									
Corrected χ^2	17.8	13.9	12.2	8.5	8.9	1.8	—		
P	<.01	<.05	<.05	<.05	<.05	NS	—		

In the United States[‡]

Year of Entry Into Study	Mean (SD) Annual Suicide Rates for Age Groups of Individual Cohorts[*] 15-19 yr	20-24 yr	25-29 yr	30-34 yr	35-39 yr	40-44 yr
1945-1949	3.8	10.3	12.8	18.9	20.4	26.1
1950-1954	3.8	11.0	15.2	18.2	22.8	
1955-1959	5.4	12.8	18.4	23.5		
1960-1964	6.6	17.0	25.1			
1965-1969	9.0	26.3				
1970-1974	11.9					

[*] Based on a mean rate per 100,000 population.
[†] ANOVA indicates analysis of variance.
[‡] Adapted from Murphy and Wetzel.[2]

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Presented at the Southern Medical Association Scientific Assembly, Atlanta, November 1986.

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TABLE 2

SUICIDE RATES FOR WHITE WOMEN IN EAST TENNESSEE AND THE UNITED STATES

In East Tennessee

Year of Entry Into Study	Mean (SD) Annual Suicide Rates for Age Groups of Individual Cohorts[*]							ANOVA Within Each Cohort as it Aged[†]	
	15-19 yr	20-24 yr	25-29 yr	30-34 yr	35-39 yr	40-44 yr	45-49 yr	Corrected χ^2	*P*
1949-1953	1.2	3.3	5.0	6.6	7.5	9.9	11.4	15.5	$<.05$
1954-1958	0.5	3.5	5.2	8.0	11.8	8.2	—	17.1	$<.01$
1959-1963	2.3	5.0	5.4	8.1	13.7	—		13.6	$<.01$
1964-1968	1.3	3.9	5.5	7.3	—			7.6	NS
1969-1973	1.6	5.5	10.0	—				9.3	$<.01$
1974-1978	4.0	6.1	—					0.3	NS
1979-1983	4.0	—							
ANOVA between cohorts at specific ages[†]									
Corrected χ^2	6.3	2.0	7.0	0.3	6.6	0.5			
P	NS	NS	NS	NS	.05	NS			

In the United States[‡]

Year of Entry Into Study	Mean (SD) Annual Suicide Rates for Age Groups of Individual Cohorts[*]					
	15-19 yr	20-24 yr	25-29 yr	30-34 yr	35-39 yr	40-44 yr
1945-1949	1.5	2.4	5.4	8.3	11.9	13.5
1950-1954	1.2	2.8	4.5	5.0	6.4	
1955-1959	1.6	4.5	7.0	9.0		
1960-1964	1.7	5.0	8.4			
1965-1969	2.6	6.4				
1970-1974	3.3					

[*] Based on a mean rate per 100,000 population.
[†] ANOVA indicates analysis of variance.
[‡] Adapted from Murphy and Wetzel.[2]

parable to the recent national birth cohort studies done by Murphy and Wetzel,[2] and Goldney and Katsikitis.[6]

Methods

The data for my study came from the Tennessee State Division for Health Statistics and refers to health service area one and two. This region is defined geographically by the Smoky Mountains on the east, the Cumberland Plateau on the west, their convergence on the south, and the Virginia border on the north. The population of this 24-county region ranged from roughly 600,000 when the study began in 1949 to roughly 900,000 when it ended in 1983. The data are not divided according to race, since East Tennessee has only 2% blacks and 1% foreign born.

In Tables 1 through 3 mean annual suicide rates were calculated using the Statistical Analysis System. The first five-year cohort consists of persons aged 15 to 19 years between 1949 and 1953 (born between 1934 and 1939). The rate for the initial cohort is located at the top left corner of the Tables. As survivors aged, the mean annual suicide rate for each successive five-year period was also calculated, giving seven time periods between 1949 and 1983 for the first cohort. The same procedure was applied to the six additional cohorts succeeding the initial group in age. For each successive cohort, there is one less grouping for which there are data, reflecting the use of the Kruskel-Wallis technique for analyzing each cohort and the changes between successive cohorts.

Results

Age cohort analysis of suicide rates in adolescents in East Tennessee reveals rates similar to the national trend, in contrast to reports that the South and rural areas as a whole have lower suicide rates.[4,5]

Comment

It has long been known that suicide varies dramatically in different areas of the world and in different areas of the United States.[3-5] It appears

TABLE 3

SUICIDE RATES FOR MEN AND WOMEN COMBINED, ALL RACES, IN EAST TENNESSEE AND THE UNITED STATES

In East Tennessee

Year of Entry Into Study	Mean (SD) Annual Suicide Rates for Age Groups of Individual Cohorts*							ANOVA Within Each Cohort as it Aged†	
	15-19 yr	20-24 yr	25-29 yr	30-34 yr	35-39 yr	40-44 yr	45-49 yr	Corrected χ^2	*P*
1949-1953	2.0	5.7	8.2	8.4	13.2	15.6	15.1	18.8	<.01
1954-1958	1.7	10.0	7.1	12.6	23.4	19.8	—	25.3	<.01
1959-1963	3.2	7.1	10.1	14.8	20.1	—		16.4	<.01
1964-1968	3.2	12.1	16.2	13.8	—			11.5	<.01
1969-1973	5.6	17.1	17.8	—				9.5	<.01
1974-1978	8.1	14.8	—					6.9	<.01
1979-1983	8.0	—							
ANOVA between cohorts at specific ages†									
Corrected χ^2	18.5	15.4	12.4	6.9	10.0	1.4	—		
P	<.01	<.01	<.05	NS	<.01	NS	—		

In the United States‡

Year of Entry Into Study	Mean (SD) Annual Suicide Rates for Age Groups of Individual Cohorts*					
	15-19 yr	20-24 yr	25-29 yr	30-34 yr	35-39 yr	40-44 yr
1945-1949	2.5	5.9	9.1	13.0	15.4	17.3
1950-1954	2.4	6.6	10.6	13.3	16.3	
1955-1959	3.4	8.4	12.6	15.9		
1960-1964	4.0	10.7	15.9			
1965-1969	5.7	15.1				
1970-1974	7.2					

*Based on a mean rate per 100,000 population.
†ANOVA indicates analysis of variance.
‡Adapted from Murphy and Wetzel.[2]

that East Tennessee is an area of higher vulnerability for suicide. This information should alert groups serving community health in this area and also challenge epidemiologists to identify cultural, geographic, biological, and other variables that may operate to influence suicide rates.

Acknowledgment:

Thanks to Janet Humphries, M.P.H., Tom Wooley, Ph.D., and David Larson, M.D., for their help with this paper.

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Preoperative Transcatheter Embolization Of an Unusual Intraorbital and Intracranial Meningioma

PETER E. DROSS, M.D.

Introduction

Preoperative embolization of intracranial meningiomas has been advocated to facilitate surgical removal by decreasing operative blood loss.[1] Both absorbable and nonabsorbable embolic agents have been used depending on the tumor location and extent of arterial blood supply. Preembolization assessment of potentially dangerous external carotid-internal carotid and external carotid-vertebral artery anastomoses is mandatory for safe embolization. The following is a description of a successful preoperative embolization of an unusual giant intraorbital and intracranial meningioma.

Case Report

An 81-year-old white woman gave a four-year history of progressive proptosis of the right eye. Physical and neurologic examination revealed mild ataxia, with cranial nerves (CN) II through XII intact.

Cranial CT showed a hyperdense, uniformly enhancing mass situated at the skull base within the right anterior and middle cranial fossae. Axial scan through the mid-orbital level demonstrates tumor extension within the lacrimal gland fossa with associated exophthalmos (Fig. 1). Sagittal reconstruction view (Fig. 2) clearly shows the degree of intraorbital and intracranial extension. Diagnostic angiography via the percutaneous femoral approach shows extensive blood supply from external carotid artery (Fig. 3) as well as tumor blush. There were no external carotid to internal carotid or vertebral artery anastomoses, and specifically no middle meningeal to ophthalmic artery interconnection. Embolization was performed via Seldinger transfemoral technique with Gelfoam (gelatin sponge) particles measuring 1 mm × 1 mm delivered into the external carotid artery using the flow-directed technique of Kerber.[2] Embolization was terminated when the tumor blush and feeding vessels could no longer be seen (Fig. 4). Following embolization, the patient did not experience neurologic deficits or cranial nerve palsies.

Surgical removal via frontotemporal craniotomy showed the tumor arising from the pterion with extension to the base of the skull and the orbit. The patient required only one unit of blood and was later discharged without complication.

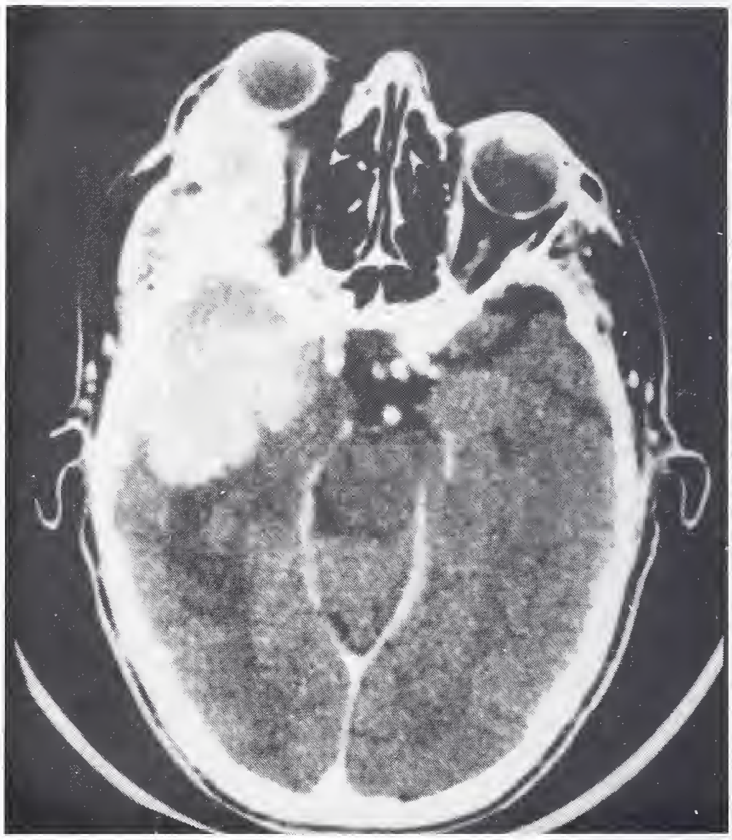

Figure 1. Mid-orbit, axial CT slice showing exophthalmos and tumor within lacrimal gland fossa.

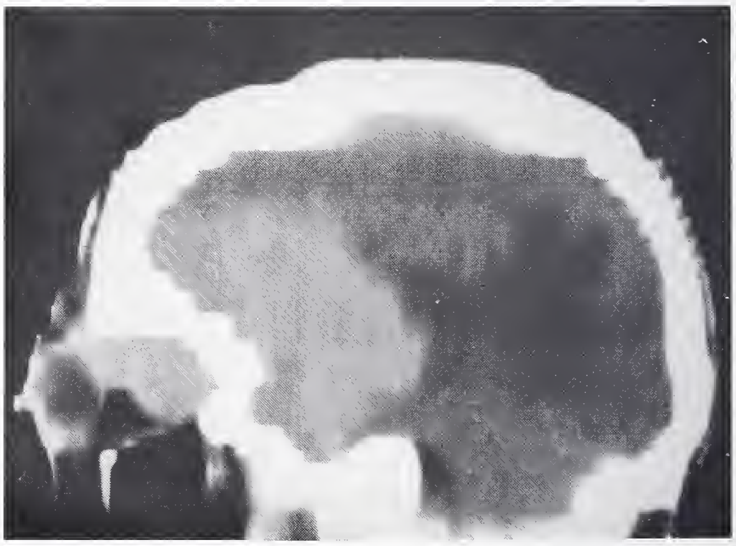

Figure 2. Sagittal reconstruction view showing degree of intraorbital and intracranial extension.

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Reprint requests to P.O. Box 862, Covington, KY 41012 (Dr. Dross).

Discussion

Preoperative embolization of intracranial meningiomas has been considered a safe preparatory step for surgical resection and can be performed during the same session as diagnostic angiography.[3] The most important factor influencing tumor recurrence is the degree of surgical resection.[4] By reducing tumor vascularity, and thus intraoperative bleeding, one can facilitate the total removal of intracranial meningiomas.[5]

Currently available occlusive materials can be divided into those that cause temporary occlusion and those that are permanent. Temporary occlusive agents include Gelfoam, microfibrillar collagen, and autologous clot, and lead to vascular obstruction in from three to seven days, after which material degradation occurs and vascular flow returns to its preembolization state.[6] These materials are best used when operative resection is to be performed within a few days following embolization, and carry a low risk of permanent damage to normal tissues. Permanent occlusive agents include polyvinyl alcohol (PVA), Silastic spheres, and bucrylate and can be used for either preoperative embolization or as the main therapeutic modality if surgery is not indicated.[6] We advocate Gelfoam, as was used in this case, whenever embolization is undertaken in vessels that have potential external carotid to internal carotid or vertebral artery anastomoses.[1]

Meningiomas best suited for preoperative embolization are highly vascular, giant meningiomas (7 cm or larger in diameter) of the skull base where surgical occlusion of the arterial supply is most difficult.[1] The most effective hemostasis can be achieved in meningiomas with primarily or exclusively external carotid supply.[7] The meningioma in this case met all these criteria. The major risks associated with embolization include neurologic deficits caused by reflux of occlusive material into the internal carotid artery and cranial nerve palsies (especially VII and XII) caused by occlusion of external carotid artery branches (especially petrous branch of middle meningeal artery supplying CN VII and neuromeningeal branches of ascending pharyngeal artery supply-

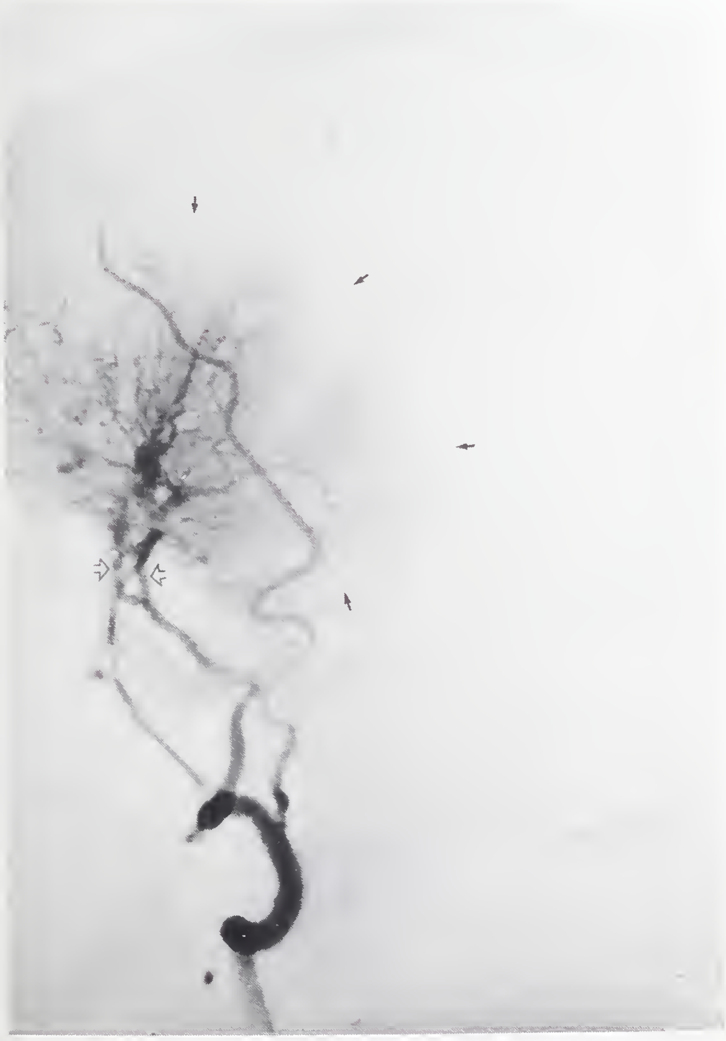

Figure 3. Preembolization, selective external carotid injection showing meningeal feeders (open arrows) and extent of tumor blush (black arrows).

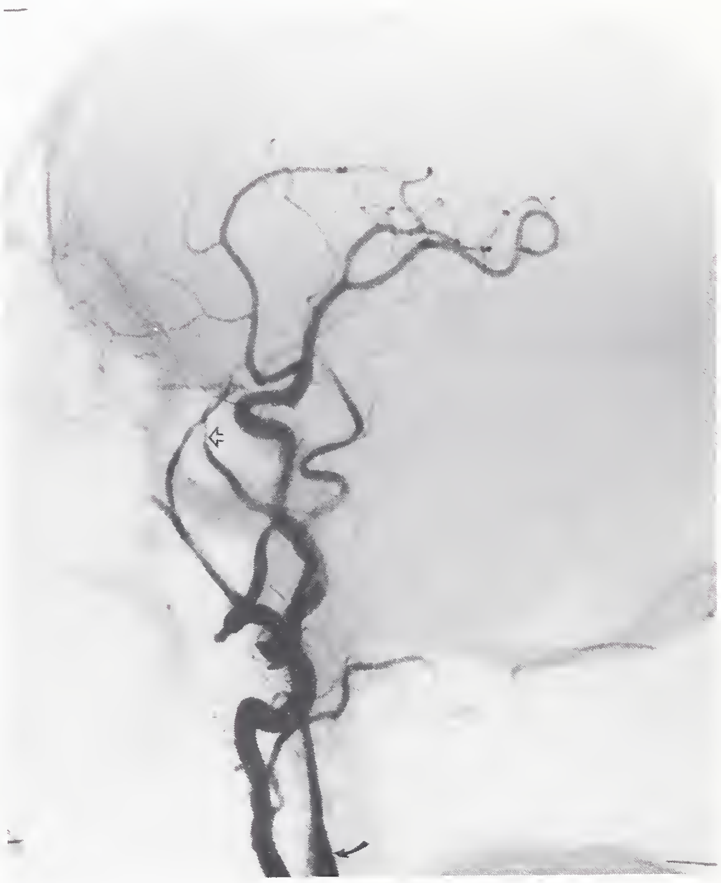

Figure 4. Postembolization selective external carotid injection showing occlusion of major meningeal feeders (open arrow) with reflux into internal carotid artery (curved arrow). Note the absence of tumor blush.

ing CN IX through XII) supplying the vasa nervorum of cranial nerves.[1,6] Potentially dangerous collaterals must therefore be assessed before safe embolization can be performed.

Intraorbital meningiomas are almost exclusively related to the optic nerve sheath[8] and therefore are unusual within the lacrimal gland fossa. The present case is most unusual, showing intraorbital extension primarily into the lacrimal gland fossa. As far as I am aware, this is the only case reported of a successful preoperative embolization of an intraorbital (lacrimal gland fossa) and intracranial meningioma.

Acknowledgments:

The author wishes to thank Stephen Natelson, M.D., of Fort Sanders Neurosurgery for referral of this case and Melanie Kelley for secretarial assistance.

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APRIL 1988

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Peritoneo-Paricardial Hernia

A Review and Case Report

RUDY T. COBB, M.D. and THOMAS V. STANLEY, M.D.

Introduction

Diaphragmatic herniation has been well documented in the literature. Traumatic herniation following blunt and penetrating trauma as well as various congenital anomalies of the diaphragm represent the basis for the vast majority of diaphragmatic hernias.[1] Traumatic hernation of abdominal viscera through the middle leaflet of the central tendon with direct entrance into the pericardial sac has been described so rarely that it has not been included in the discussions on diaphragmatic hernias in the standard textbooks of surgery.[2] The combined series of Harrington and Hedblom include 1,251 diaphragmatic hernias with no mention of any peritoneo-pericardial hernia. Smith and Lippert found 14 cases of isolated traumatic peritoneo-pericardial herniation reported in the world literature in 1957.[3] A review by Alvarez et al[4] in 1983 revealed only 20 cases of isolated peritoneo-pericardial communication through a defect in the diaphragm secondary to trauma to which he added a case.

Case Report

A 64-year-old black man was referred for evaluation of epigastric pain and possible gastric obstruction. He experienced severe cramping epigastric pain with immediate vomiting after any attempt at oral intake, but otherwise was comfortable. He complained of occasional dyspnea while reclining, but that readily resolved upon assuming the upright position. He had been treated with digoxin and furosemide for many years because of his symptoms and cardiomegaly demonstrated radiographically, although his symptoms remained unchanged. Eighteen years prior to this admission he sustained an extensive right chest wall contusion, multiple rib fractures, and a myocardial contusion with subsequent pulmonary complications. Eight years later, he was hospitalized for evaluation of epigastric cramping pain and was found to have a loop of large bowel in his chest. He declined surgical intervention as his symptoms totally resolved with nasogastric suction.

Physical examination on this admission revealed a large well-nourished man in no distress. His pulse was 88/min with a blood pressure of 140/100 mm Hg. Jugular venous distention was not appreciated, and the lungs were clear bilaterally. The cardiac point of maximum impulse could not be localized, and the heart sounds were diminished.

The abdomen was obese with normal bowel sounds and moderate tenderness on deep palpation in the epigastrium. The hematocrit was 41% and the WBC count was 10,500/cu mm including 80% neutrophils and 11% lymphocytes. An EKG showed a rate of 83/min with a left anterior hemiblock. A chest x-ray demonstrated multiple old rib fractures and an air-fluid level overlying the heart (Fig. 1).

The cardiac silhouette was very large, with smooth, indistinct borders. The lateral chest view showed the heart to be displaced posteriorly by a mass, and a barium enema revealed a loop of colon overlying the heart shadow. An upper GI series verified a total gastric obstruction, with a small portion of gastric cardia tapering off in the direction of the abnormal air space overlying the pericardium.

The patient was taken to the operating room with the preoperative diagnosis of incarcerated diaphragmatic hernia. A subclavian venous catheter was inserted, revealing a central venous pressure reading of 23 cm of water. He was then placed in a partial right lateral decubitus position and through a left rectus-splitting incision the abdomen was entered; the total absence of omentum and transverse colon was the most striking finding. A small bulb of stomach was identified by palpation of the nasogastric tube. The spleen was shifted to the midline and was adjacent to the diaphragm. A tear in the central tendon was found with omentum adherent to the ring of the defect.

With division of the omental adhesions, the transverse colon, the entire omentum, and the distal stomach were returned from the chest to the abdomen. There were no adhesions to the pericardium and no evidence of a hernia sac. The heart was readily visible, and digital examination within the pericardium did not demonstrate a pleuropericardial defect. A 6 × 4-cm defect was easily repaired with a two-layer closure using permanent suture material, and the pericardium was drained using closed suction drainage through an extraperitoneal tube. With all the reduced viscera being viable, the abdomen was closed, and when the patient was returned to the supine position, an immediate central venous pressure reading of 0 cm of water was obtained. His postoperative course was complicated by the development of bilateral lower lobe atelectasis. He was discharged on his tenth postoperative day with a follow-up chest x-ray showing a normal cardiac silhouette (Fig. 2).

Discussion

This case illustrates many of the characteristic features of a traumatic peritoneo-pericardial hernia. The sequence of events is so similar to previous case reports that a characteristic pentad pattern can be established. When the patient sustains severe trauma, an occult diaphragmatic in-

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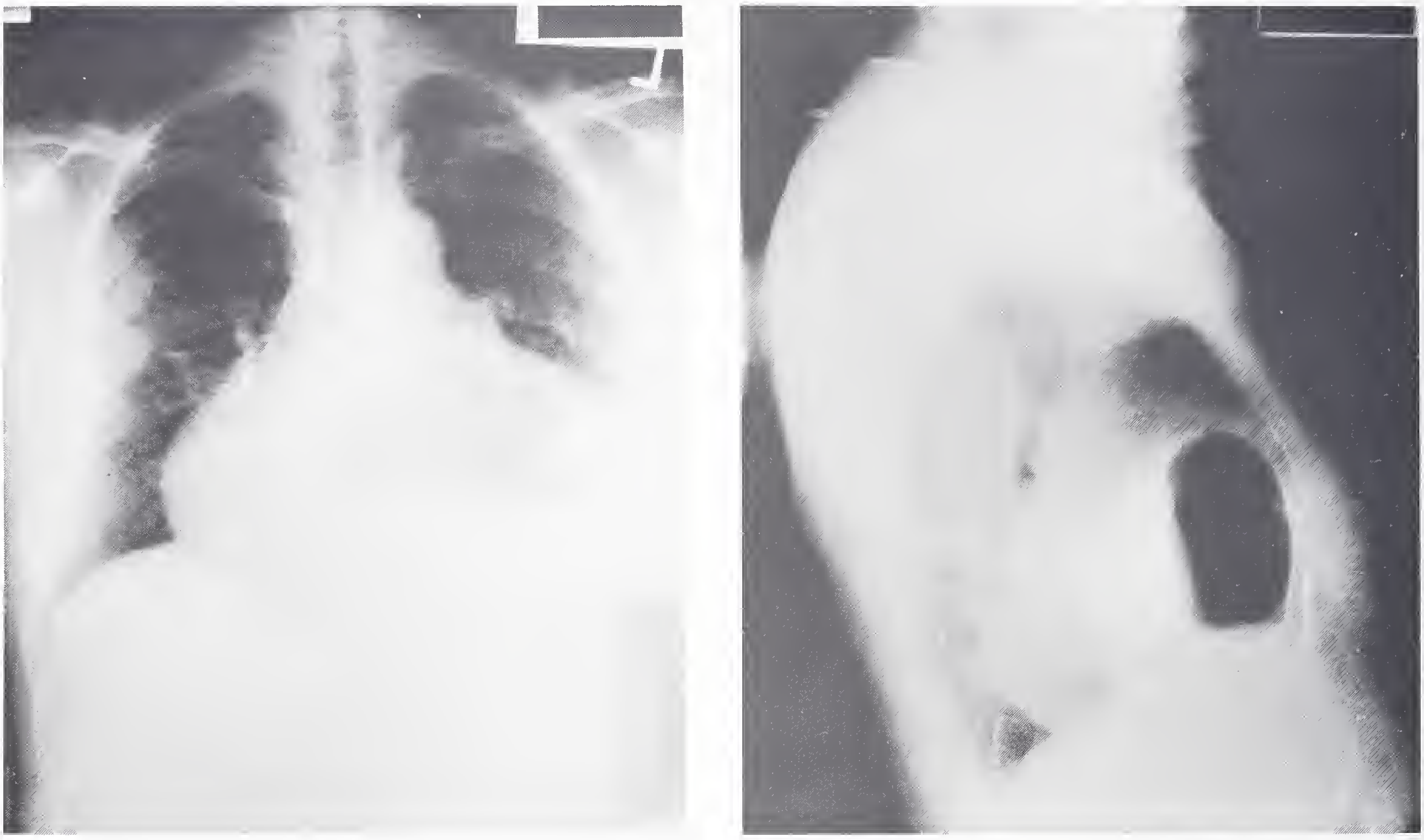

Figure 1. Preoperative PA and lateral chest x-ray.

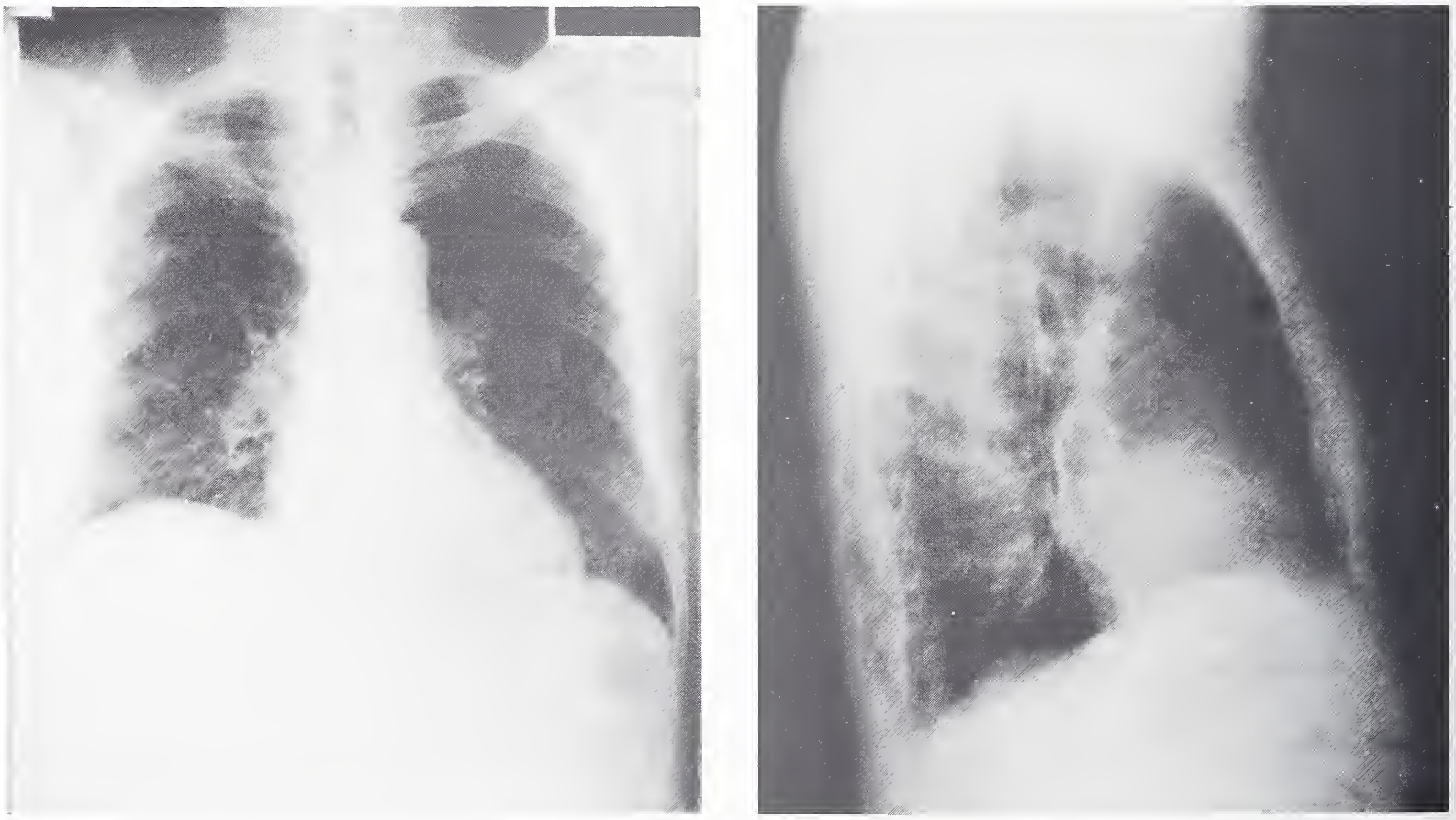

Figure 2. Postoperative PA and lateral chest x-ray.

jury is overshadowed by associated major injuries; after an asymptomatic period of variable duration, the injury is manifested by the gradual or abrupt onset of debilitating cardiac or gastrointestinal symptoms. Complete resolution follows surgical reduction of the incarcerated contents and repair of the hernia defect.

The embryology and anatomy of the diaphragm are of significance in the congenital and acquired types of hernia, but are of little value in predicting the location of a traumatic defect, which does not necessarily conform to the sites of potential weakness. About 95% of the traumatic diaphragmatic hernias involve the left side of the diaphragm, possibly because the splinting action of the liver helps to reduce the frequency of diaphragmatic disrupton on the right side; when they are encountered, however, right-sided defects are very large.[5]

Herniation of abdominal contents through a diaphragmatic defect does not always immediately follow the injury, and concomitant injuries may distract the examiner from recognition of diaphragmatic injury. A fractured pelvis is seen in over half of the cases of blunt injury associated with diaphragmatic disruption, and splenic injury is frequent.[6] When a small diaphragmatic defect is plugged with omentum, the margins of the defect are prevented from healing, leaving the site vulnerable to future stress.[5] As with the more common peritoneo-pleural traumatic hernias, the lapse of time from injury to diagnosis of peritoneo-pericardial hernia is frequently quite variable, and has been reported to range from just hours to as long as 23 years, with an average time of seven years.[7]

The signs and symptoms appear as various viscera pass through the diaphragmatic aperture. Dyspnea, cyanosis, palpitations, and chest pain may easily pass for symptoms of myocardial disease.[3] When viscera become incarcerated, signs and symptoms of intestinal obstruction accompany ischemic gastrointestinal obstruction.

Pathophysiologically, herniation of abdominal viscera into the pericardial cavity affects cardiac dynamics, with a subsequent decrease in cardiac output, to the extent that 80% of the patients develop symptoms suggesting cardiac disease.[8,9]

Treatment consists of surgical reduction of the herniated viscera, with repair of the defect. Our review revealed that a transthoracic approach is more frequently reported, primarily because the correct postoperative diagnosis of a peritoneo-pericardial hernia is lacking, and a hernia through the left leaf of the diaphragm is mistakenly expected.[2,10] There are several reports that the inital use of a transthoracic incision was found to be inadequate for proper repair when the correct diagnosis was finally established intraoperatively, and extension of the incision into the abdomen was required. We find no report that a transabdominal approach alone was inadequate when the correct preoperative diagnosis was established. Thus, it is imperative that the distinction between a chronic peritoneo-pleural hernia, for which a transthoracic approach is indicated, and a chronic peritoneo-pericardial hernia, for which a transabdominal approach is indicated, be established preoperatively. Although adhesions to the heart within the pericardium have not been reported as a problem, some surgeons still adamantly insist that a thoracotomy is the only logical approach.[3] In view of the increased morbidity, the increased chances of phrenic nerve injury, and the frequent report of inadequate exposure using a thoracic incision, it is our conclusion that an abdominal incision is the approach of choice for the repair of a peritoneo-pericardial hernia.

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Blunt Injury to the Diaphragm

D. PRESTON SMITH, M.D. and KIMBALL I. MAULL, M.D.

Blunt injury to the diaphragm is most commonly caused by deceleration (falls, motor vehicle crashes) and compression (crush injury, blasts) of the trunk. Although the true incidence of diaphragmatic injuries from blunt trauma is unknown, it is reported to be between 2% and 4% in patients with multisystem trauma.[1] Diaphragmatic injury adds an increased risk of death but may be easily missed.

Case Report

A 16-year-old white boy was involved in a two vehicle crash in which his vehicle, traveling approximately 30 to 40 mph, was struck broadside on the left by a truck traveling at about the same speed. He was an unrestrained left rear-seated passenger and sustained blunt trauma to the chest and abdomen. He was initially treated at a local hospital, where an initial chest roentgenograph showed a left hemothorax; it was treated by tube thoracostomy, and the patient was transferred to the University of Tennessee Medical Center at Knoxville via Lifestar air ambulance. Upon arrival the patient was in acute respiratory distress. His blood pressure was 95/60 mm Hg, pulse 120/min, and respirations over 30/min; breath sounds were markedly decreased on the left. Chest tube output was approximately 300 cc. The abdomen was scaphoid. The left wrist and shoulder were tender and swollen. Initial arterial blood gas determinations showed pH 7.30, P_{CO_2} 42, and P_{O_2} 119 on oxygen supplemented face mask ventilation. Microscopic hematuria was present. Repeat chest film was diagnostic of diaphragmatic laceration with herniation (Fig. 1).

The patient was taken immediately to the operating room for exploratory celiotomy and repair of the left diaphragm. Prior to intubation, a right thoracostomy tube was inserted. A large amount of blood was noted in the abdominal cavity, and the stomach, spleen and portions of the colon and small bowel were in the left hemithorax. After they were replaced in the abdomen, the spleen was found to have a type IV laceration involving the hilum. A splenectomy and repair of a 10-cm diaphragmatic laceration were performed. There were no other intra-abdominal injuries found.

The patient was successfully extubated on the first postoperative day. The right and left thoracostomy tubes were successfully removed on the fifth and sixth postoperative days, respectively, and the patient was discharged home on the ninth postoperative day.

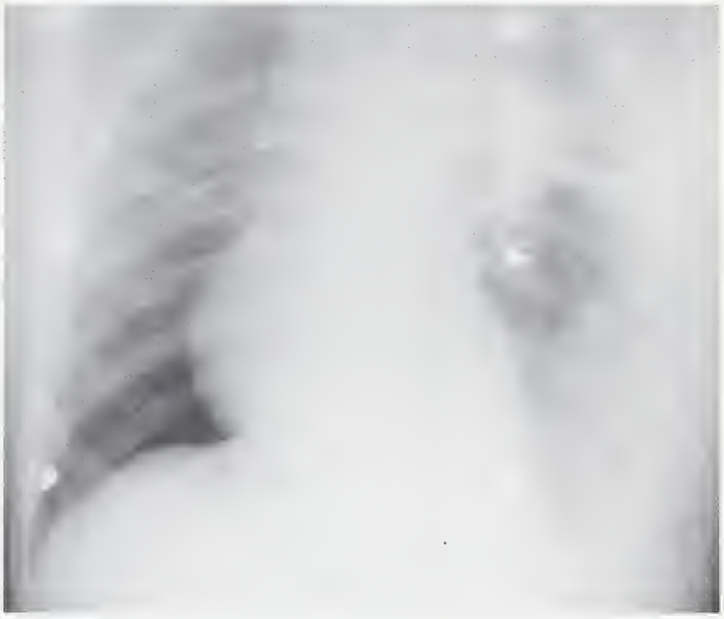

Figure 1. Chest film demonstrating large air shadow above ruptured left diaphragm with nasogastric tube coiled within. Left diaphragm is not seen, while right side is sharply defined. Note right pneumothorax.

Discussion

Diaphragmatic injury must be considered in any victim who has sustained significant blunt injury. The mechanism of injury in diaphragmatic rupture is controversial, but it is believed that the focus of impact is initially in the abdomen and is transmitted to the diaphragm. The explosive rise in intra-abdominal pressure causes radial tears in the posterolateral aspect of the hemidiaphragm corresponding to the point of embryologic fusion of the leaflets. The left hemidiaphragm, once thought to be involved in more than 90% of blunt diaphragmatic trauma, is more commonly injured but with less disparateness. The liver buffers the right hemidiaphragm against injury, but right-sided lacerations still occur and may be related to three-point restraint use.[2] On the left side, diaphragmatic herniation of the stomach, colon, small intestine, spleen, and omentum oc-

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curs due to the relative negative intrathoracic pressure.

The clinical presentation of patients with diaphragmatic herniation varies. In the acute phase, signs and symptoms are directly related to the effects of abdominal viscera within the pleural cavity. For example, if substantial herniation occurs early, respiratory compromise results, a pathophysiologic effect not unlike tension pneumothorax. If laceration occurs without substantial herniation, a latent phase may occur, sometimes lasting several years, during which the patient occasionally experiences episodic obstructive symptoms. The diagnosis may be made serendipitously or ultimately because of intestinal obstruction. This phase is marked by the development of acute abdominal pain which may lead to intestinal gangrene if untreated. Obviously, diagnosis at initial presentation is to be encouraged. The physician must be aware of the injuries most commonly associated with diaphragmatic rupture due to blunt trauma, namely fractured ribs, fractured pelvis, ruptured spleen, and closed head injury.

The diagnosis requires a high index of suspicion, especially when other more apparent injuries are present. Decreased breath sounds on the ipsilateral side is the most frequent physical finding and is directly related to the herniation of abdominal contents. Basilar dullness, mediastinal shift, dyspnea, hypotension, and audible bowel sounds in the hemithorax are reported, and a scaphoid abdominal configuration is suggestive. These signs, in association with abdominal pain and evidence of external trauma to the left side of the thorax or to the abdomen, should alert one to the diagnosis.

Though the chest roentgenograph is the most frequent and important method of diagnosing diaphragmatic rupture, the initial chest x-ray may be interpreted as normal in 40% of cases. Four radiographic findings suggesting diaphragmatic hernia are described: (1) archlike shadows resembling a high diaphragm, (2) extraneous shadows or gas bubbles extending above the diaphragm, (3) shift of the mediastinum, and (4) disk or platelike atelectasis above an archlike shadow.[3] Other radiologic findings include an indistinct hemidiaphragm and fractures of the lower ribs. The presence of a nasogastric tube extending above the left hemidiaphragm is diagnostic. Contrast studies, fluoroscopy, computed tomography, and sonography may be helpful in demonstrating diaphragmatic herniation.[4] Peritoneal lavage may return no effluent. Conversely, it may be diagnostic if a chest tube is present on the affected side and lavage fluid returns through the tube.

Although the definitive treatment of a ruptured diaphragm is surgical repair, opinions differ regarding the operative approach. Most trauma surgeons recommend celiotomy for acute diaphragmatic injuries unless there are suspected life-threatening intrathoracic problems. When this approach is used, reduction and diaphragmatic repair can be easily accomplished and associated injuries dealt with. Thoracotomy is useful for diaphragmatic herniation that is recognized late, since many such patients have extensive adhesions in the region of the diaphragm and to the lung that make repair through the abdomen difficult. On the right side, a thoracoabdominal approach may be needed, and regardless of the side of injury it is prudent to prepare for possible thoracotomy before attempting celiotomy.

Summary

Diaphragmatic injury must be considered in all victims of blunt trauma; it is easily missed if not considered by the clinician. Chest x-ray may be either diagnostic, abnormal, or normal. If it is normal, the injury may not be recognized initially in some patients, and a latent phase may ensue, which may lead to intestinal obstruction and/or respiratory compromise. Early operation is curative.

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VUH Morning Report

Pancytopenia in a Patient Previously Treated for Hodgkin's Disease

Case Report

A 53-year-old white man with a history of Hodgkin's disease was admitted to Vanderbilt Hospital for fever and pancytopenia. Hodgkin's disease, stage III A mixed cellularity, had been diagnosed four years earlier when he was seen for right inguinal adenopathy. A splenectomy was performed and the patient received eight cycles of chemotherapy with nitrogen mustard, vincristine, prednisone, and procarbazine (MOPP), as well as extensive periaortic and right inguinal node irradiation. He had a complete remission, and was thought to be cured. He did well until five days prior to admission when he developed fever, pain, and swelling of the right knee. Arthrocentesis by his local physician showed cloudy fluid with 3,000 RBCs/cu mm and 21,000 WBCs/cu mm with a predominance of "abnormal" monocytes. A peripheral CBC showed a WBC count of 3,900/cu mm (normal 5,000 to 10,000) with a differential of 46% lymphocytes, 23% "atypical" monocytes, 28% monocytes, and 3% segmented neutrophils. His platelet count was 21,000/cu mm (normal 150,000 to 350,000) and his hematocrit 26% (normal 42 to 50). A bone marrow biopsy on hospitalization showed "myelodysplasia," and he was transferred to Vanderbilt Hospital.

On transfer, he had a temperature of 103°F orally, but his vital signs were stable. Though he appeared uncomfortable, his physical examination showed only swelling and tenderness of his right knee. Laboratory examination showed an SGOT of 91 IU/L (normal 4 to 40), alkaline phosphatase of 805 IU/L (normal 40 to 110), and an LDH of 389 IU/L (normal 125 to 250). CBC showed a WBC count of 4,000/cu mm with 4% segmented neutrophils, 70% lymphocytes, and 26% monocytes. Hematocrit was 21% and platelet count 49,000/cu mm. Therapy with antibiotics was begun, and blood cultures subsequently grew *Pseudomonas aeruginosa*. Bone marrow biopsy was repeated and showed acute myelomonocytic leukemia. The pain and swelling of his knee were believed due to leukemic infiltration of the joint. Repeated arthrocentesis yielded negative cultures and no crystals. The patient was subsequently treated wtih cytarabine and daunorubicin.

Discussion

Reports noting the development of second malignancies after successful therapy for Hodgkin's disease are becoming increasingly common in the medical literature,[1] perhaps because successful therapy is available for Hodgkin's disease and a large number of patients are surviving for long periods of time.[2] The role of various factors leading to the development of second malignancies is uncertain, but a correlation seems to exist between the development of a second malignancy and the intensity of therapy, especially if both chemotherapy and radiation are used.[1] Both hematologic and nonhematologic tumors are seen with increased frequency following the treatment of Hodgkin's disease. Acute nonlymphocytic leukemia is most common. The risk of acute nonlymphocytic leukemia in patients who have had intensive alkylating chemotherapy and irradiation combined may be as high as 1,000 times that in the normal population. Chemotherapy alone is associated with a lower risk of acute leukemia, while those treated with irradiation alone rarely develop leukemia.[2]

Acute leukemia following therapy for Hodgkin's disease has certain characteristics; often there is a period of preceding pancytopenia or dyserythropoiesis, and chromosomal aberrations are common when karyotyping is done. There is usually refractoriness to therapy, with treatment resulting in partial or complete remission in only 25% of cases.[3]

There appears to be a two- to three-fold increase in the occurrence of other malignancies following treatment for Hodgkin's disease, for which radiation and chemotherapy seem to be equally responsible.[2]

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Jaundice in a Middle-Aged Man

Case Report

A 56-year-old white man was admitted to Vanderbilt Hospital for evaluation of two weeks of painless jaundice; he had had a similar episode in 1979, when abdominal ultrasound had revealed gallstones for which he underwent a cholecystectomy. No stones were found other than a few small soft ones in the gallbladder but an intraoperative cholangiogram showed slight narrowing of intrahepatic ducts and one constricted segment in the common duct. The liver appeared slightly fibrotic but biopsy revealed only cholestasis. At that

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Prepared by Jason Morrow, M.D., Hugh J. Morgan chief medical resident, Vanderbilt Medical Center, Nashville.

time his serum bilirubin, which had been 12 mg/dl (normal 1 to 1.2) on admission, returned to normal after surgery. The patient did not seek medical treatment again until 1984, when screening blood work revealed a gamma glutamyltranspeptidase (GGT) of 946 IU/L (normal 11 to 63), alkaline phosphatase of 139 IU/L (normal 20 to 125), SGOT of 93 IU/L (normal 0 to 50), and serum bilirubin of 1.8 mg/dl (normal .1 to 1.2). CT scan of the abdomen revealed mild fatty liver infiltration. A liver-spleen scan was normal, and antimitochondrial antibody was negative. Primary sclerosing cholangitis was considered as a diagnosis, but because the patient was asymptomatic it was decided not to treat him. He continued to do well until two weeks prior to admission when the painless jaundice began, with light stools, dark urine, and abdominal swelling.

He was referred to Vanderbilt, where examination revealed marked jaundice and ascites. His liver was not palpable but his spleen could be felt 3 cm below the left costal margin. Laboratory findings were SGOT 104 IU/L, alkaline phosphatase 344 IU/L, and serum bilirubin 23 mg/dl. CT scan of the abdomen revealed ascites, a small, nodular liver, and a large spleen. Endoscopic retrograde cholecystopancreatography revealed multiple strictures throughout the common bile duct, with pruning of the intrahepatic radicals typical of sclerosing cholangitis (Fig. 1). Colonoscopy revealed a normal large bowel. The patient's ascites was treated with spironolactone. Liver transplant is being considered.

Discussion

Primary sclerosing cholangitis (PSC) is a chronic inflammatory disorder affecting the intrahepatic and extrahepatic bile ducts; its etiology is not known. It has been associated with diverse diseases, most commonly ulcerative colitis.[1] It may have an autoimmune basis, and has been associated with certain HLA types, most commonly HLA B8 and DR3. Portal viral and bacterial infections have been considered possible causes.[1]

Clinically, PSC follows a progressive course. Men in the third to fifth decades are most commonly affected, with symptoms including right upper quadrant abdominal pain, icterus, pruritis, and weight loss, but many patients are asymptomatic.[2] Diagnosis depends primarily on the demonstration of radiologic abnormalities of the bile ducts.[2] Demonstration of segmental strictures of the biliary tree by endoscopic retrograde cholecystopancreatography or transhepatic cholangiography is diagnostic in 80% of cases.[1] Liver biopsy is usually not helpful; it may reveal cholestasis, portal inflammation, fibrosis, or cirrhosis. Laboratory tests classically show a cholestatic picture, with increased GGT, alkaline phosphatase, and bilirubin.

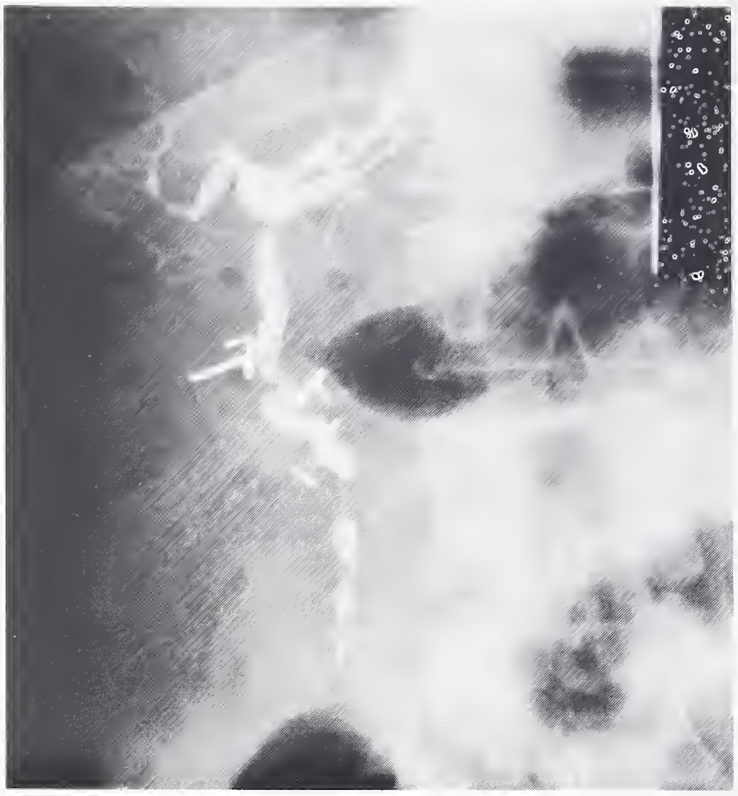

Figure 1. Endoscopic retrograde cholecystopancreatography showing strictures throughout the common bile duct and pruning of intrahepatic radicals.

Differential diagnosis of PSC includes bile duct carcinoma, anomalies of the biliary tree, primary biliary cirrhosis, and scarring from previous surgery.[1] Prognosis is generally poor. End stage liver disease usually develops within a few years after diagnosis, and death is usually due to a complication of liver failure. Treatment with a number of agents, including steroids, azathioprine and cholestyramine, have met with limited success, as have surgical procedures.[2,3] Liver transplant may be a therapeutic option.[1]

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Tennessee AIDS Advisory Committee

FREDIA S. WADLEY, M.D., MSHPA

On Sept. 3, 1987, twenty-seven individuals appointed by Commissioner James E. Word met for the first time as Tennessee's AIDS Advisory Committee. The members, who represent various groups, disciplines, and organizations with an interest in AIDS, received their charge to review the issues and make recommendations concerning the direction Tennessee should take to prevent the spread of the HIV (human immunodeficiency virus) infection, and to provide appropriate health, social, and support services for those with the infection.

Three subcommittees (Policies, Services/Support, and Prevention/Education) have been established in order to effectively cover more issues. Although the committee will meet through February or March 1988, it has been encouraged to prioritize issues and make a preliminary report by the first week of November 1987. Recommendations needing legislative attention this session would need to be submitted by then.

As of Aug. 31, 1987, there had been 260 cases of AIDS reported in Tennessee since 1981. Although this sounds minor compared to New York's almost 11,000 reported cases, we must consider a few figures to understand the concern.

- For every case of AIDS, we may have ten cases of AIDS-Related Complex (ARC). This would mean over 2,500 ARC cases in Tennessee.
- It is estimated that 12,000 to 15,000 individuals are infected with HIV in this state.
- According to predictions of the Tennessee Communicable Disease Control Division, based on the AIDS trend in Tennessee, by 1991 we will have 5,000 cumulative AIDS cases reported since 1981, up to 2,500 newly diagnosed AIDS cases that year alone, and 50,000 cumulative ARC cases.

The best we could hope for for 1991 is that only 1,500 to 2,000 AIDS victims will need medical care in Tennessee. A current conservative estimate for care is $40,000 per case, which still adds up to a cost of $60 million to $80 million for Tennessee AIDS patients in 1991. While definitive information on the extent to which Medicaid pays for services to AIDS patients is lacking, the federal Health Care Financing Administration has estimated that at any one time, 40% of AIDS patients receive services paid for by the Medicaid program.[1 (p 127)]

There are at least five characteristics of HIV infection that make it a difficult public health problem with which to deal. First is the long asymptomatic phase during which a person is infectious, as well as the unique feature of being infectious for life. There is no practical definitive test for the virus that can be used in screening and clinical settings. The major two routes of transmission being sexual (especially homosexual) activity and intravenous drug use has definitely decreased the enthusiasm of many people for allocating already limited resources to AIDS prevention or care. There is no known cure or vaccine, and virtually all AIDS patients die. The confidentiality and discrimination problems surrounding the infection, and the intense fear generated in some people by the infection, coupled with the perception that all who have it are homosexuals or intravenous drug users, has presented unprecedented discrimination problems. Health professionals have become so afraid of discrimination problems that they have not felt comfortable in responding as they would like.

One of the hottest policy issues relative to AIDS hit Tennessee in August as children returned to school. Some parents were demanding that health and education officials either guarantee that there was absolutely no chance of their children getting the HIV infection from a classmate, or exclude HIV infected students from

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From the Tennessee Department of Health and Environment, Nashville. Dr. Wadley is chief medical officer of TDHE.

school. Since this same problem has occurred in various states, three things have become apparent:

- Teachers, students, and parents who have prior information about the infection have fewer reservations, and are more comfortable when an HIV infected student arrives for classes.
- Education is definitely helpful, but some individuals will never change their minds regardless of the amount of information presented. Either they don't believe the health officials giving the facts, or they don't want to accept even an exceptionally small risk.
- In previous court cases and rulings, a general policy to exclude HIV infected students has not been upheld.

With intensive case investigations from 1981 until now, it still stands that the three ways that the HIV infection is transmitted are sex, blood, and birth. The recent reports that 12 health care workers have converted to HIV antibody-positive status have alarmed the health industry and the general population. Although this finding should be given serious regard, we should also remember that of over 2,400 health care workers being followed because of HIV exposures through needle sticks or significant blood and body fluid contaminations, only 12 have been known to convert to a positive test (nine due to needle sticks and three to mucous membrane or skin exposure to blood).[2] During these studies, it has also become apparent that the HIV infection is not as easily transmitted as hepatitis B through a needle stick exposure. Though this is good news for the optimist, the pessimist restates it that not everything is known about the virus, and this lack of knowledge precludes acceptance of the statement that transmission does not occur by casual contact.

While precautions should be taken by health care workers liable to exposure to HIV infected blood, the question being asked now is whether there is a sufficient risk of blood exposure in school or the workplace to warrant excluding the infected student or employee from the premises.

The New York State Senate Majority Task Force on AIDS summarized the legislative dilemma by saying:

> The AIDS epidemic presents state lawmakers with the challenge of protecting the public health while maintaining a legal tradition that includes a commitment to civil liberties, due process and equal protection under the law. Constitutionally, this commitment requires that policy be based upon reason and the relevant medical evidence rather than on fear and prejudice. In the case of AIDS, any new laws must follow reliable medical evidence.[1](p 47)

Physicians across the state are being asked questions by individual patients or community groups concerning AIDS. If the Tennessee Department of Health and Environment can be of assistance in providing you with facts and figures concerning HIV infection, please call our Communicable Disease Control Division (615) 741-7247. If you have any recommendations that you would like the Advisory Committee to consider, please send them to me: Fredia Wadley, M.D., Tennessee Department of Health and Environment, 344 Cordell Hull Bldg., Nashville, TN 37219-5402.

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Loss Prevention Case of the Month

The Emergency Room Zoo

J. KELLEY AVERY, M.D.

Case Report

About 6 PM one Saturday evening, a healthy-appearing man was brought to the very busy emergency room (ER). He was obese, extremely strong, extremely combative and difficult to control, and required multiple attendants to hold him on the stretcher and eventually required restraints. On admission to the ER his blood pressure was 120/50 mm Hg, pulse 120/min, temperature 99.4°F, and respiratory rate 36/min. Drug overdose was suspected, and a drug screen was ordered. The patient was retained in the ER and monitored at irregular intervals. About 7½ hours after admission to the ER, a drug screen was reported, and the salicylate level was reported as 100. The junior house physician, who was working in the ER that evening, did not recognize the significance of the high salicylate level and was, therefore, not as aggressive as he might have been in the management of his patient, but he did contact the attending physician on two occasions and was advised to order blood gases, which was done; in spite of his obvious confusion as to the problem with his patient, the attending physician did not come to the ER to assist in the management of this very difficult patient.

Again there was significant delay from the time the blood gases were ordered and the report was received in the ER. At about the time the report was made, the patient suddenly began to convulse and died.

A lawsuit was brought, charging the resident, the attending physician, and the hospital with negligence in the care of this patient. A sizable settlement was negotiated on the part of all parties.

Loss Prevention Comments

It is difficult to exonerate the first year resident, who admitted he did not know the full significance of the salicylate level. Certainly, by that time in his medical education he should have known that such a level was extremely dangerous. To his credit, he was very busy, running from room to room to attend to many more or less minor complaints. Of course, since the attending physician is responsible for the "supervision" of the housestaff, it was his duty to personally involve himself in the management of the patient.

Complicating the problem was a laboratory that somehow sent the first report to the wrong area of the hospital, so that consequently it was 7½ hours before the report arrived in the ER. In the ER, on a busy weekend night, it is easy to see how a patient might "get lost" in the rush; systems should be in place to track the laboratory work and to follow up if the report has not been received within the allotted time. The report of the blood gases ordered by the attending physician was also delayed.

This extremely unfortunate and tragic accident appeared to be the result of the confluence of activity of a junior house officer who knew less than he should have, an attending physician who felt that his resident knew more than he really did, and the lack of prompt and efficient systems to expedite the reporting of laboratory work, once accomplished.

Dr. Avery is chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood, and medical director for Ambulatory/Outpatient Services, St. Thomas Hospital, Nashville.

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president's page

JAMES T. GALYON

Medical Care for the Indigent

Medical care for the indigent has always been a problem for Tennessee physicians. This has not changed. The Governor placed this issue high on his priority list when he was running for office. He has already acted in two ways, first, to establish a commission to study the problem and to recommend long range solutions, and second to assist in some creative financing. Some hospitals contribute funds that will insure even greater federal matching funds; they will then be returned to these same hospitals as Medicaid reimbursement—a sort of bureaucratic "casting of bread upon the waters." Though this is very innovative and helps to some extent, most of us realize it will have no long range impact.

Levels of Medicaid reimbursement are so low that it is difficult to have a large percentage of such patients and still deliver quality care. This leads physicians to refuse to routinely accept Medicaid patients for treatment, driving them to institutional care (i.e., medical school clinics, public health clinics, hospital clinics, emergency rooms). They may also be driven to so-called Medicaid mills, which survive by delivering care to a large volume of patients, sometimes without regard to its quality.

Pediatricians and primary care physicians have made inroads into this situation by establishing "homes" for children's medical care. This is a place where the child must go for his primary medical care, and it will be accepted by Medicaid at an increased level of reimbursement.

Attempts are now being made to establish such primary care "homes" for indigent adults. The results will need close observation to see if they are successful. These are the correct trends, with more realistic reimbursement to keep the medically indigent patient in the mainstream of medicine.

Hospitals have been the focus of considerable media attention because of the problems of indigent care. Some believe this is entirely the problem of public hospitals, supported by tax dollars. Others believe all hospitals have an obligation to care for the medically indigent. Obviously, someone pays for uncompensated care. If not the taxpayer, then it is the paying patient or his insurer.

In Memphis, charity has reared its head again. A concerned and dedicated physician has started a new outpatient clinic supported by churches, private individuals, business, and industry. Maybe the word charity can be allowed back in our vocabulary again.

There will never be any single answer to medical care for the indigent. Multiple solutions must be tried; these should include a more realistic reimbursement to physicians to keep the indigent in the physician's office where the care is best and the cost is economical.

If government at all levels, federal, state and local, really believes medical care for the indigent is a legitimate concern of theirs, then they will have to ante up to pay for these services—not just promise a lot and pay a little.

Finally, a little charity by all of us, as always, goes a long way in helping those less fortunate.

James T. Galyon, M.D

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NOVEMBER, 1987

editorials

Thanksgiving Day— Of, By, and For the People

The very efficient managing editor of this publication, who rides herd on me and other errant authors and keeps things in proper order, called yesterday and asked if I was working on another editorial. I told her I was thinking about it. She allowed as how, since I always write a Thanksgiving editorial, she knew that I was. I wasn't. I am now, but you can tell it is in a rather desultory fashion. After all, it's only a couple of weeks since Labor Day, we have Halloween yet to go, and I bet I haven't gotten more than half a dozen Christmas catalogs yet. So it's hard to get in the mood for Thanksgiving, what with fall starting only this morning.

While it may be hard to get in the mood yet for the capital "T" Thanksgiving, it isn't for the small "t" one. In fact, I stay that way. I'm like the after dinner speaker who said, "I'm happy to be here with you this evening," and after he had thought a moment, continued, "Hell, I'm happy to be anywhere." I *am* thankful to be where I am, and doing what I'm doing. I enjoy practicing medicine, despite its tribulations (which are perhaps more niggling but certainly less grave than they were 50 years ago, when I finished high school and started up the ladder). I am thankful for good health, a lovely wife, and a fine family, and while I might not have all the creature comforts I can imagine, I do have most of what I want, and certainly a lot more than I need. But those are things for which we are to give thanks daily to the One who the apostle Paul said can supply your every need through His great riches in glory. (Of course Paul was talking about things other than creature comforts, since he was writing to the Philippians from jail.) Thanksgiving Day is about something else.

Though a day of Thanksgiving to God, and even the ancient and not so ancient gods, permeates mankind's history generally, as well as our own, Thanksgiving Day as a national holiday was first proclaimed by President Lincoln during the War between the States to give thanks for this nation, the United States of America, which were then anything but united.

Nashville has just gone through an incredibly tasteless, unconscionably expensive mayoral campaign in which the two candidates evenly divided four million dollars for the privilege of impugning each other's morals, integrity, responsibility, and taste. It reminded me of schoolyard jousts in which one would hurl an insult and his opponent would shout back, "You're another." Or "I'm rubber and you're glue. Everything you say to me bounces back and sticks on you." And so on. Since we had to have a runoff election, Nashville citizens have been subjected to that sort of nonsense since last spring. Everyone was so thoroughly sick of it that more than one yard sported a sign proclaiming "Nobody for Mayor." A large percentage of the votes cast were negative ones.

But who would have it otherwise? Though certainly one would wish for more restraint and less expense, the percentage of the world's population with access to a free electoral process is small, and so therefore is that price we pay to maintain ours. The real culprits are the media, particularly television, which provides such a ready platform for the gauche antics of the candidates and their supporters.

In an address before the House of Commons shortly after the Second World War, Winston Churchill said, "No one pretends that democracy is perfect or all-wise. Indeed, it has been said that democracy is the worst form of government except all those other forms that have been tried from time to time." The freedom of elections was wrested from tyrants at great cost, and its survival is tenuous at best, depending as it does on the whimsy of a fickle citizenry. The birth of this nation followed a long, painful delivery in which the survival of the offspring was frequently in doubt. It suffered many pangs during its adolescence, and in its adulthood its wisdom and effectiveness have often been questioned. There are those who now see signs of senility.

No nation or government is immortal. At Gettysburg Mr. Lincoln asked that this nation under God have a new birth in freedom. Rebirth must happen with each generation, as a nation survives only so long as it lives in the hearts of its countrymen. There are many who would remove it from God's providence; that would be the seeds of its undoing. Thanksgiving Day was ordained and constituted not for bellies stuffed with turkey and minds jellied by football; its observance is a moral obligation, "that government of the people, by the people, and for the people should not perish from the earth." The threat is not from without, but from within; the Civil War ended, but the battle for survival never does.

J.B.T.

Happy
Thanksgiving

Human Resources

An easily ignored communication made it across a synapse even as the paper it was printed on drifted toward my round file. The words that demanded attention said "Georgia Department of Human Resources." Well, resources for what?

Words play tricks on you—or more precisely, the use of words can play tricks on the unwary. People can use words to play tricks on you, too, but that is not what I'm talking about. I'm talking about words used imprecisely inadvertently—through ignorance or carelessness.

We have a cat. She is female. Because I said "she" you would have known our cat was female even if I had not said so. But if I had said, "We have a female," you would have said, "A female what?" *N'est-ce pas?* Female is an adjective, you say? It can apply to anything that lives, you say? Then why do you say (if you do say—if you do not say, you are in a distinct minority—a *correct* minority), then why do you say, "A 38-year-old female presented to the emergency room . . ."? A female what? That has become a more or less standard way to open a case presentation. (In fact, it is wrong twice. One can present oneself to the emergency room, but one cannot present *to* anything. The word present is a transitive verb, which means it requires an object—a *direct* object. So much for the grammar digression.)

Now back to females and males. There are boars and sows; there are mares and stallions; there are bulls and cows (there are geldings and steers, too, which are neither, and shoats and children, which are both, but we aren't talking about them; they aren't applicable to this act). There are also men and boys and girls and women (Vive la différence!). To refer to them as males and females lumps them into a glob of living beings that includes not only the animal kingdom (except for the likes of steers and earthworms), but much of the plant kingdom (or the higher parts of it) as well.

To call a man or boy a male, or a woman or girl a female, dehumanizes them. It is the trend. If that's all we are, then I suppose it is correct to refer to us as *resources*—or also vice versa, I guess. It depends upon how we are viewed, and by whom.

Except on the highest philosophical plane, throughout history one has had to be a king (or high priest, or what-not) to achieve much status as an individual. The only religions to teach the value of the individual got around it by pragmatically considering equality as in the sight of God, since "here on earth have we no continuing place." Kings got that way by divine right (after they got that way by being either stronger or smarter or craftier). The serfs, and also the freeman (not to mention the nobles), were their "human resources" that (not who) brought in all their other resources. The nobility had each his own human resources. The rest had not much—human or otherwise.

In penning the words "all men are created equal," Mr. Jefferson et al were obviously not implying that each individual is as strong or as smart or as crafty as any other. They knew better than that, and so did—and does—everybody else (I think). They were espousing a new—and refreshing—concept that no one is anybody else's resource, and that the individual is more important than any collection of individuals (i.e., the state), and also that the function of the group (i.e., the state) is solely to maintain the individual's life and liberty and to allow that individual to pursue happiness.

To start with, way back yonder (*way* back yonder) there were only individuals. They found it hard to survive that way, and so they banded together. Someone found out, say, that a stick extended the arm, and with it you could strike down your enemy (who was anyone who wanted—or had—what you wanted) before he could reach you. Everyone else shouted, "He ought'a be king," and so he was. Israel wanted a king like everybody else. God said He had always thought He was their king, but if that was the way they wanted it, that's the way they would have it. Look what happened to Israel. Man (Israel is just an example) traded his individuality for security and uniformity, and reaped conformity; he became a resource.

To refer to the United States government as the federal government is both accurate and inaccurate. According to the Constitution, the United States is a federation of sovereign states, and its constitutionally granted powers and prerogatives are strictly limited. It appears though that from a practical standpoint once a man has ever gotten to Washington he has ceased being a federalist and became a nationalist. The first state to test the glue was Massachusetts; it was rebuffed by none other than our own Andrew Jackson, who as president threatened armed intervention if Massachusetts tried to secede. Massachusetts backed down, but South Carolina did not. After five bloody years the federation (or union) died, and from its ashes rose the nation. It has been strengthened by every Supreme Court chief justice, each of whom has progressively eroded the prerogatives of the states, gathering momentum through the years. What we have in actuality is a national, and not a federal, government.

The government has not (and indeed could not have, of course) done all of this in a closet. It has all been done with the help and concurrence, and indeed the encouragement, of the citizenry, which has not only assented, but has urged the "federal" government to assume the lead. "Only the federal government has enough money (or enough power) to. . . ." And where does the federal government get the money and the power? From its citizens.

It gets the money and the power from its human resources—its males and females. We have come full circle, and joined the club.

J.B.T.

Life in a Putty Knife Factory

A book editor of *JAMA* once wrote that people do not borrow books; people steal books. I might quibble a little with that, in that stealing implies acquiring someone else's property illegally and without permission. I think therefore that to say people steal books is, at least for the most part, hyperbole. Nevertheless, it is true that many, many books take up permanent residence with others than their legal owner. It is a pity, because though I do not often need to look anything up in the book of the same name as this piece, it is a reference work of sorts, and I used to have it. Though I do still own it, I don't have it anymore.

To refer to *Life in a Putty Knife Factory* as a reference work could also be considered hyperbole, I guess—in fact, is. It was written by a clown named H. Allen Smith, who was an observer and chronicler of life on the local scene, largely in the New York City of the Forties and Fifties (of this century); his theme was the ridiculous, of which the Big Apple, which it was not called then, had, as it still has, a plenty. What I wanted to look up was the preface, which is a single sentence quote from somebody to the effect that the world is like a factory given over entirely to the manufacture of putty knives.

Though that too is hyperbole, at times I think it is not much.

The last time I thought of that book was one morning about a year ago while I was sitting in the gallery of the Senate Chamber in our nation's capitol. They were making putty knives that day, as they (both Houses) have often done both before and since. I would not be unwilling to allow them that privilege if their activities did not require that so much of my own time be relegated to that same pursuit. As it is, I resent it.

There are a great many things that the Congress could profitably spend its time on if they could only lay politics aside and get on with it. On the other hand, perhaps it is just as well, as they would likely only screw them up, too. Everybody has difficulty seeing past AIDS right now; it does cast a long, dark shadow, but there are other problems that are at least as serious, which in our preoccupation we need to not lose sight of.

We have in the *Journal* this month a study of suicides in East Tennessee, which shows an increase among adolescents, contradicting conventional wisdom that suicide, and especially adolescent suicide, is low in rural areas, and particularly in southern rural areas. It isn't. In a White Paper on Adolescent Health, the AMA notes that 5,000 young people under 19 commit suicide each year, and 50,000 attempt it; both morbidity and mortality for adolescents on all counts is 11% higher today than 20 years ago. Though suicide is a factor, it is only one; it ranked fourth as a problem among Tennessee teenagers in a survey conducted by the TMA Auxiliary (TMAA). The others in order of priority were alcohol, other drugs, teen pregnancy, and depression.

The same white paper notes that two-thirds of American youth have used an illicit drug by the time they finish high school, and one in five smokes cigarettes daily; one in 16 drinks alcoholic beverages daily, and 41% report having consumed five or more drinks at least once. Eighty percent of deaths in the 15- to 19-year age group are caused by accidents, and this same group is responsible for a third of all violent crimes.

Six hundred thousand teenage girls and half that many boys work as prostitutes; their average age is 15. The highest rate of many sexually transmitted diseases occurs in sexually active teenagers, and health professionals believe they will be the next population group to be seriously affected by the acquired immune deficiency syndrome (AIDS). Teenage mothers account for 46% of births to unmarried women and one-third of all abortions; maternal mortality in 15 year olds is two and a half times that in women over 20.

This is a real, true horror story, which, along with the AMA, our TMAA is trying to address. The TMAA has taken this on as a project, and this March sponsored a highly successful Teen Health Workshop at Memphis State University involving 478 high school students and advisors from the western region of Tennessee, conducted by concerned, knowledgeable professionals. It is projected as an annual affair, and in 1988 moves to the University of Tennessee at Chattanooga, where 750 participants are expected. In sponsoring this effort, your spouses are performing a real service to Tennessee's youth, and reflect credit on our TMA members.

I commend to your attention the communication from Mrs. Gayanne Burns, president of TMAA, carried elsewhere in the *Journal,* in which she reports on the Auxiliary's activities. Sadly, and inexplicably, membership in the Auxiliary is declining. Perhaps it is because of a perception that the Auxiliary does nothing more significant than making putty knives. I assure you there is more. I urge you to urge your spouse to add her (or his, as appropriate) efforts to the efforts of all those others who among other things are working toward bettering the health of our future citizens. Nothing is more important than that.

J.B.T.

Drug Abuse

To the Editor:

For some time we have been involved in drug and alcohol abuse in the Memphis area, and one area in particular continues to be a problem. The combination of "T's and Blues" has continued despite its decreased significance with the advent of cocaine. It would seem to me that one reasonable way to eliminate this problem altogether would be to cease production of either of the two drugs involved, or make them unusable for this type of drug abuse. Pentazocine (Talwin) is now marketed in the United States as Talwin-NX and contains Narcan which should greatly reduce the street value of this as an abused drug. Unfortunately, it seems that the NX product is not approved in Canada, and Talwin is still available in its original form in that country. Since Talwin seems to be a good pain reliever, and seems to have considerable use for that problem, perhaps we should consider the other component—tripelennamine (Pyribenzamine), or as it is better known, PBZ. I have asked many physicians in the Memphis area, including some of my colleagues who have been practicing for considerably more years than I, if they have ever written a prescription for Pyribenzamine. Most physicians will answer the question about the drug with another question—"What is Pyribenzamine?" In fact, many of the drug addicts in this area oftentimes will not buy a "set of T's and Blues" unless the drug has the Geigy label on it. It's interesting that they sometimes mispronounce the name as "Gag-it," but the presence of the label is still quite important to the addict.

Since there seems to be very little use for it as an antihistamine in this region of the country, could we perhaps ask our colleagues throughout the state, both in medicine and pharmacy, to consider what might be an appropriate usage for the drug? If each of us were, for instance, to discuss the problem with our own Geigy Pharmaceutical representative (detail person), perhaps the company would consider removing the product from the market, or certainly curtailing its availability.

Although the forces of the marketplace certainly should determine to some extent the availability of any prescription medication, perhaps some additional consideration should be given to these products to eliminate, or curtail, their abuse. Could I ask your help, or your response to the problem?

Robert D. Kirkpatrick, M.D.
210 Jackson Ave. #503
Memphis, TN 38105

in memoriam

Jesse E. Adams, Jr., age 61. Died September 3, 1987. Graduate of Harvard Medical School. Member of Chattanooga-Hamilton County Medical Society.

Gerald M. Halprin, age 50. Died August 27, 1987. Graduate of Wayne State University College of Medicine. Member of Nashville Academy of Medicine.

William J. Sheridan, age 87. Died August 20, 1987. Graduate of University of Michigan Medical School. Member of Chattanooga-Hamilton County Medical Society.

Willard O. Tirrill, Jr., age 82. Died September 22, 1987. Graduate of Vanderbilt University School of Medicine. Member of Nashville Academy of Medicine.

TMA Members Receive AMA Physician's Recognition Award

Twenty-five TMA members qualified for the AMA Physician's Recognition Award during August 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Harry Baer, M.D., Nashville
James B. Bell, M.D., Seymour
Marian L. Bertotti, M.D., Johnson City
John W. Brock, III, M.D., Nashville
Lloyd R. Broomes, M.D., Murfreesboro
Susan H. Bryant, M.D., Nashville
Benjamin F. Byrd, M.D., Nashville
Frederick J. Chapin, M.D., Cookeville
Robert T. Doster, Jr., M.D., Nashville
Deborah R. Doyle, M.D., Nashville
Roy C. Ezell, M.D., Nashville
Jeffrey C. Fosnes, M.D., Centerville
George J. Grasel, M.D., Union City
Saul A. Juliao, M.D., Nashville
Phillip R. Langsdon, M.D., Memphis
Raymond F. Mayer, M.D., Memphis
Douglas P. Mitchell, M.D., Nashville
V. Tupper Morehead, M.D., Nashville
William W. Potter, III, M.D., Morristown
Carolyn C. Price, M.D., Memphis
Norman S. Propper, M.D., Kingsport
Burton M. Rudolph, M.D., Knoxville
Jack C. Sanford, Jr., M.D., Memphis
S. Steve Snow, M.D., Nashville
Brent A. Soper, M.D., Madison

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY
James E. Abell, M.D., Chattanooga
Richard G. Alvarez, M.D., Chattanooga
Swarna K. Bareddy, M.D., Chattanooga
Valerie A. Boaz, M.D., Chattanooga
Luther E. Chandler, M.D., Hixson
Gussie E. Hawkins, M.D., Chattanooga
William M. Kittle, III, M.D., Chattanooga

Jack B. McCallie, M.D., Chattanooga
Jeffry B. Potash, M.D., Chattanooga
Walter B. Rose, M.D., Chattanooga
Larry A. Sargent, M.D., Chattanooga
Ralph A. Sellers, II, M.D., Chattanooga
Vinay Vermani, M.D., Chattanooga
Christopher R. Yetter, M.D., Chattanooga

CONSOLIDATED MEDICAL ASSEMBLY OF WEST TENNESSEE
William H. Woods, Jr., M.D., Jackson

DICKSON COUNTY MEDICAL SOCIETY
Rolland Luplow, M.D., Dickson

KNOXVILLE ACADEMY OF MEDICINE
Larry C. Brakebill, M.D., Knoxville
Sarkis J. Chobanian, M.D., Knoxville
Deborah J. Christiansen, M.D., Knoxville
J. William Earnhardt, M.D., Knoxville
Aseed Etezadi-Amoli, M.D., Knoxville
Randal O. Graham, M.D., Knoxville
Joseph L. Thompson, III, M.D., Knoxville
Stephen G. Wilson, Jr., M.D., Knoxville

LAKEWAY MEDICAL SOCIETY
C. Cole Anderson, M.D., Morristown

MEMPHIS-SHELBY COUNTY MEDICAL SOCIETY
Michael Batson Brady, M.D., Memphis
James Lee Ellis, Jr., M.D., Memphis
Phillip Edward Green, M.D., Memphis
Thomas Arthur Hughes, M.D., Memphis
Leslie R. McGowan, M.D., Memphis
John Pervis Milnor, III, M.D., Memphis
Michael Scott Muhlbauer, M.D., Memphis
Carol Ann Phillips, M.D., Memphis
Robert Allen Price, M.D., Memphis
Douglas Lynn Rose, M.D., Memphis
Hanson Philip Sachs, M.D., Memphis
David Dale Sloas, M.D., Memphis
Tolan Hoang Vu, M.D., Germantown

NASHVILLE ACADEMY OF MEDICINE
John Roger Clark, M.D., Brentwood
David Nando Jones, M.D., Nashville
Edwin Jacob Lilly, M.D, Nashville
Larry M. Mitchell, M.D., Nashville
Loren Douglas Nelson, M.D., Nashville
William Faxon Payne, M.D., Nashville
Lawrence Warren Travis, M.D., Nashville

SUMNER COUNTY MEDICAL SOCIETY
John G. Bartek, M.D., Gallatin
Robert T. Mitchell, M.D., Gallatin

WILLIAMSON COUNTY MEDICAL SOCIETY
Thomas Augustus Jantz, M.D., Franklin

personal news

John W. Johnson, M.D., Memphis, has been certified as a diplomate of the American Board of Anesthesiology.

John W. Zirkle, M.D., Jefferson City, has been recognized for Advanced Achievement in Internal Medicine. The AAIM examination is a new voluntary three-module examination that recognizes "certified internists who, in times of rapid change and expansion of medical science, continue to maintain the knowledge required to provide excellent care."

announcements

CALENDAR OF MEETINGS

NATIONAL

Dec. 3-6	Eye-Bank Association of America—Boston
Dec. 3-6	Society for Ear, Nose and Throat Advances in Children—Fairmont Hotel, Denver
Dec. 6-9	American Epilepsy Society—Baltimore
Dec. 10-13	American Academy of Psychoanalysis—Scottsdale, Ariz.
Jan. 10-13	Inter-American Society for Chemotherapy—Clearwater, Fla.
Jan. 18-24	Southern Clinical Neurological Society—Fort Myers, Fla.

TENNESSEE MEDICAL ASSOCIATION
153RD ANNUAL MEETING
April 13-16, 1988
Peabody Hotel, Memphis

Update '87

The TMA Auxiliary appreciates the continued support of the TMA, and I take this opportunity to report our activities and plans.

AMA-ERF remains of primary importance. We believe Tennessee's medical schools and medicine in Tennessee are the winners when the TMAA leads the nation in contributions. With the continued use of the Christmas Sharing Card and smaller fund-raisers, we are hoping for 100% participation from our counties this year. Last year, we were short only two.

During the first week in March, TMA and TMAA will sponsor *two* Teen Health Workshops—one in Chattanooga and one in Memphis. Both will deal with the positive aspects of our youth and be presented in a very upbeat manner. Chattanooga has had tentative acceptances from two major celebrities to participate.

We have added a statewide emphasis on organ donation. Mrs. Milli Yium (Jackson) will be speaking to at least five or our county auxiliaries across the state as the TMAA's representative. We are making materials and information available upon request.

This year, Tennessee became the first state to appoint a resident/intern spouse to our board. Marcy Walker (William C.) of Memphis is a dynamic, very intelligent young lady who is making contact with each of our medical school deans in order to give us an increased opportunity to introduce and recruit these young people to TMA Auxiliary.

Our fall board meeting will no longer be just that. We have changed the name to Update '87 and invited all TMAA members to attend. It will be held Sept. 20-21 at the Vanderbilt Plaza Hotel. We have four major outside speakers, whose topics include "Teen Suicide," "Superwoman Syndrome," "AIDS," and "Changes in Medicine." We will hold workshops for county presidents, presidents-elect, treasurers, and AMA-ERF and membership chairmen. IMPACT-AMPAC will be emphasized. An "Idea Fair" has been organized for the first time; the concept is for counties to share their projects through individual displays. I am excited about this change, and look forward to our Update '87.

We will be sending 14 county presidents-elect to Leadership Confluences in Chicago.

All of this is on the *state* level, and we recognize that the majority of our projects are initiated on the county level. Hours of commitment in our communities are obvious only to them.

The TMA Auxiliary has a *major* problem—*membership!* We have lost members the last two years. We must put some of our energy into altering this. Many of our leaders have attended membership workshops at AMAA; we have come home with many suggestions, and will implement some this year. The membership chairman and I have prepared a brochure that will be distributed at Update '87. Hopefully, this tool will assist county chairmen in encouraging their members to join TMAA. We are asking for "Supporting" members in addition to "Participating" members (the dues are the same).

I am asking the members of TMA to lend us a hand; please encourage your spouses to join the Auxiliary (an Auxilian must be the spouse of a TMA member).

Gayanne Burns, President
TMA Auxiliary

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Please notify us six weeks in advance.

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Send to: TMA, 112 Louise Ave., Nashville, TN 37203

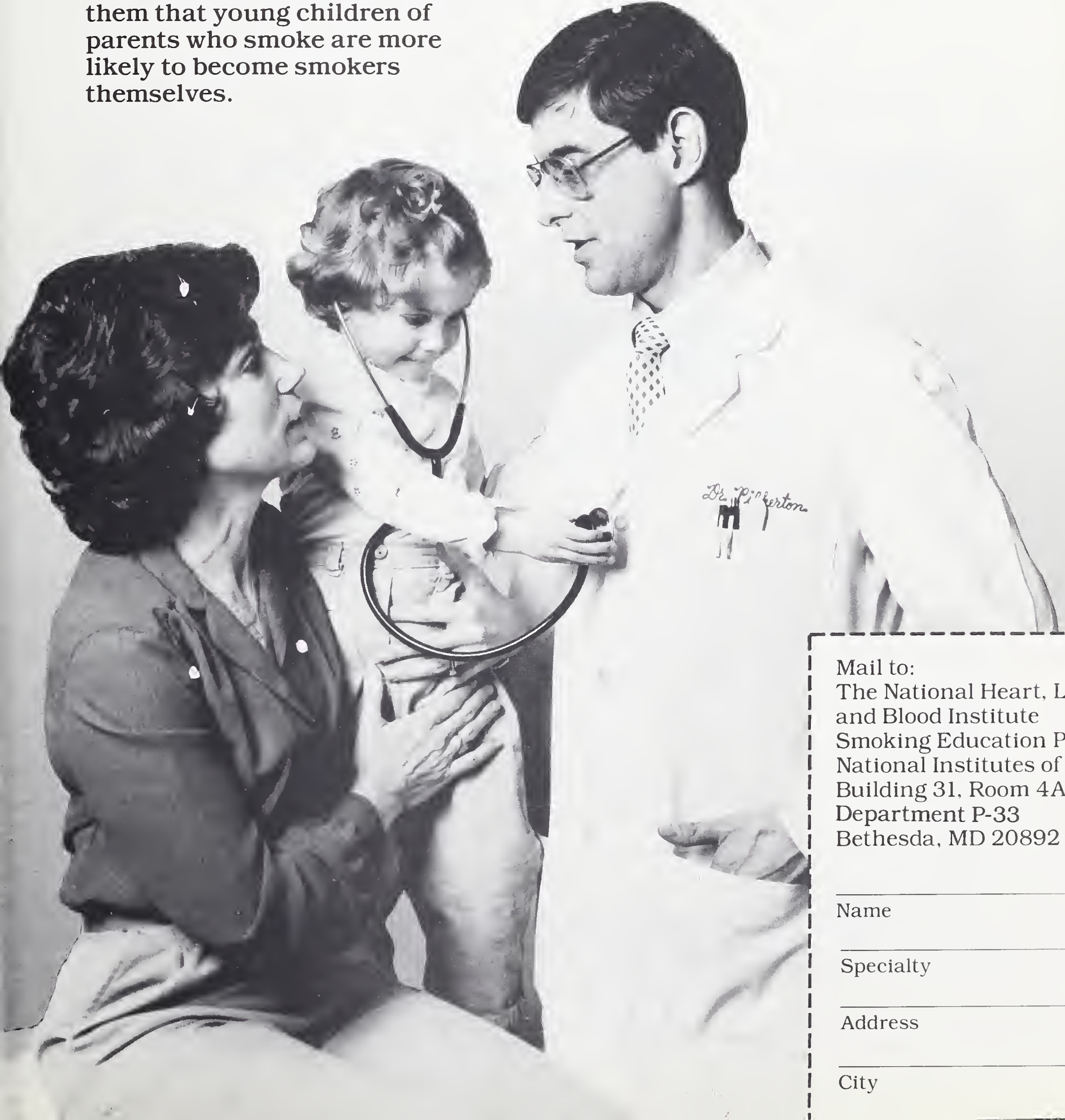
A Clinical Opportunity for Smoking Intervention

You can play a special role in reaching smokers. Encouraging parents not to smoke can improve the health of the entire family.

Take a few minutes to explain that children of parents who smoke are often more prone to respiratory infections. Tell them that young children of parents who smoke are more likely to become smokers themselves.

The minutes you spend can make a difference now, and in the years ahead.

For a free copy of *Clinical Opportunities for Smoking Intervention: A Guide for the Busy Physician*, complete the form below.



Mail to:
The National Heart, Lung,
and Blood Institute
Smoking Education Program
National Institutes of Health
Building 31, Room 4A 18
Department P-33
Bethesda, MD 20892

Name

Specialty

Address

City

Minutes of the Tennessee State Board of Medical Examiners Meeting

April 14-15, 1987

Members Present:	Duane C. Budd, M.D., President William W. Cloud, M.D., Secretary Paul Johnson, M.D., Consultant I. Lee Arnold, M.D. Phillip W. Hayes, M.D.

The meeting was called to order at 8:30 AM, April 14, 1987, by Duane C. Budd, M.D., president.

The minutes from the Dec. 16 and 17, 1986, meeting were approved as submitted.

Applications. Reciprocity applications for Drs. **Humberto Rodriguez, Kevin A. Skinner, Jerry B. Williams, Linden C. Fernando, Melinda Thompson** and **T. Allen Polk** were unanimously approved. The reciprocity application for Dr. **Robert S. Diamant** was approved with the stipulation he continue with TMA's Impaired Physician Program for one year. He may come back before the Board at the end of one year to request lifting of this restriction. The reciprocity application for Dr. **Allick M. Dube** was approved contingent to receiving a letter of recommendation from the chief of his residency program. The reciprocity application for Dr. **Nicholas N. Dzebolo** was deferred until criminal charges are cleared up. The reciprocity application for Dr. **Jorge Francheschi-Zambranan** was denied because he did not have an ECFMG certificate. The reciprocity application for Dr. **Timothy R. Oesch** was approved pending submission to a psychiatric evaluation with evaluation being satisfactory to the Secretary of the Board. The reciprocity application of Dr. **Ajay Sobti** was tabled since he was not present. The reciprocity application of Dr. **Mohammad Yaseen** was approved pending receipt of a letter of recommendation from the chief of his residency program when he is within four months of completing his third year residency, provided letter is satisfactory to the Secretary of the Board. The reciprocity application of Dr. **Louis W. DeInnocentes** was deferred pending receipt of more information concerning the malpractice suits filed against him.

The reinstatement applications of Dr. **Shivender S. Deol** and **Robert F. Berson** were tabled since they were not present. The reinstatement applications of Drs. **Andrew O. Jensen** and **Mary Ward** were unanimously approved.

The Respiratory Care Rules were unanimously approved with one amendment.

Dr. Phillip Hayes volunteered to proctor the June FLEX examination all three days.

The grandfather clause for x-ray operators was discussed. Dr. Budd will check with former Board members, Drs. Burkhart, Foreman and Ingram, as to the Board's intent concerning this subject when the original rules were promulgated. There is a difference of opinion as to whether the grandfather clause covers all x-rays that the operator had performed for at least ten years or whether it only covers chest and/or extremities.

A letter from Humana Hospital concerning notice of Board actions against physicians being made available to the chief of staff or executive director of the hospital(s) where the physician has privileges, in a timely manner, was presented to the Board. The Board's decision was that the minutes are made available to Tennessee Medical Association for publication in the *TMA Journal* and the hospitals should have these journals available to them. No further notification is planned at this time.

Hearings

Notice of charges had been previously filed against Dr. **C. M. Baylor** (see Minutes of Dec. 16 and 17, 1986). Since no further complaints have been filed against Dr. Baylor, the Board unanimously approved the dismissal of charges.

Dr. **Norman Saliba** was charged with unprofessional, dishonorable, or unethical conduct; gross malpractice, in a pattern of continued or repeated malpractice, ignorance, negligence or incompetence in the course of medical practice; habitual intoxication or personal misuse of any drugs or the use of intoxicating liquors, narcotics, controlled substances or other drugs or stimulants in such manner as to adversely affect the person's ability to practice medicine; dispensing, prescribing, or otherwise distributing any controlled substance or any other drug not in the course of professional practice, or not in good faith to relieve pain and suffering, or not to cure an ailment, physical infirmity or disease; dispensing, prescribing or otherwise distributing to any person a controlled substance or other drug if such person is addicted to the habit of using said controlled substances without making a bona fide effort to cure the habit of such patient; dispensing, prescribing, or otherwise distributing any controlled substance or any other drug to any person in violation of any law of the State or of the United States of America; and engaging in the practice of medicine when mentally or physically unable to safely do so. The Board found Dr. Saliba guilty of all charges except (1) gross malpractice, or a pattern of continued or repeated malpractice, ignorance, negligence or incompetence in the course of medical practice; and (2) dispensing, prescribing or otherwise distributing to any person a controlled substance or other drug if such person is addicted to the habit of using said controlled substances without making a bona fide effort to cure the habit of such patient. The Board suspended Dr. Saliba's license for a minimum of one year. He is to reinvolve himself with the TMA Impaired Physician Program and meet requirements for continued advocacy of that Program. He may come back before the Board at the end of one year to request lifting of suspension.

The hearing for Dr. **Bobby Watts** was continued from the Feb. 3 and 4, 1987 meeting. The Board found Dr. Watts guilty of gross malpractice, or a pattern of continued or repeated malpractice, ignorance, negligence or incompetence in the course of medical practice; dispensing, prescribing, or otherwise distributing any controlled substance or any other drug not in the course of professional practice, or not to cure an ailment, physical infirmity or disease; and dispensing, prescribing or otherwise distributing to any person a controlled substance or other drug if such person is addicted to the habit of using controlled substances without making a bona fide effort to cure the habit of such patient. The Board suspended Dr. Watts' license for a minimum of six months during which time he must attend certain continuing education courses as specified by the Board in order to update his education and to train him in certain drug usage and the potential of addiction. Dr. Watts is also required to surrender his DEA license indefinitely.

There being no further business, the meeting was adjourned at 4:00 PM.

AIM HIGH

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AIR FORCE

TMA continuing medical education opportunities

The continuing medical education accreditation program of the TMA has full approval by the Accreditation Council for Continuing Medical Education. An accredited institution or organization may designate for Category I credit toward the AMA Physician's Recognition Award those CME activities that meet appropriate guidelines. If you wish information as to how your hospital may receive accreditation, write: Director of Continuing Medical Education, Tennessee Medical Association, 112 Louise Ave., Nashville, TN 37203.

IMPORTANT NOTICE

Published in this section are all educational opportunities which come to our attention which might be of interest to our membership. As some of these are very long, full year schedules, and others are detailed descriptions of courses, in order to conserve space, most of them will be published in only one issue of the Journal.

IN TENNESSEE

VANDERBILT UNIVERSITY

Clinical Training Program

Opportunities for advanced clinical education for physicians in various subspecialties have been developed by the School of Medicine and the Division of Continuing Education of Vanderbilt University. The practicing physician, with the guidance of the faculty from the participating department, can plan an individualized program of one to four weeks to meet recognized needs and interests. The experience may include contact with patients; interaction with clinical and full-time faculty, housestaff, and fellows; participation in clinical rounds and diagnostic procedures. There is access to the Medical Center Library with excellent resources. Experience in more than one discipline may be included if time permits.

Eligibility: Licensed physicians in active practice are eligible to apply. *Credit:* AMA Physician's Recognition Award (Category 1) and/or AAFP Continuing Education Accreditation. *Application:* For information and an application contact Marilyn D'Asaro, Division of Continuing Medical Education, Vanderbilt University School of Medicine, CCC-5326 MCN, Nashville, TN 37232, Tel. (615) 322-4030.

Continuing Education Schedule

Dec. 3-5	Lasers in Otolaryngology—Head and Neck Surgery
Dec. 4-5	High Risk Obstetrics Seminar and the Everett M. Clayton-John Zelenik Memorial Lecture
Dec. 5-6	Annual Update in Anesthesiology
Jan. 30-Feb. 5	Advances in Medicine—Snowmass Village, Colo.
Feb. 13-19	Advances in Infertility and Reproductive Endocrinology—Snowmass Village, Colo.
Feb. 13-19	Practical Aspects of Diagnostic Radiology/Medical Imaging—Snowmass Village, Colo.
March 5	Early Intervention in Acute MI
March 12-19	The Spine: Current Concepts—Maui, Hawaii
March 17-18	6th Annual Tennessee Conference on Perinatal and Neonatal Care
March 28-29	Tissue-Integrated Prostheses: Aspects of the Prosthodontic Part
April 22-23	Annual Barney Brooks Lecture and the H. William Scott Society
May 6-15	Diving Medicine 1988: Medicine in the Aquatic Environment—Fiji
May 17-22	5th Annual Family Medicine Review
May 27-28	11th Annual Sonography Symposium
June 9-10	2nd Annual Neonatology Conference
June 15-19	Contemporary Medical Imaging V—Destin, Fla.
July 18-22	11th Annual Contemporary Clinical Neurology—Hilton Head Island, S.C.

For information contact Division of Continuing Medical Education, Vanderbilt University School of Medicine, CCC-5326 MCN, Nashville, TN 37232, Tel. (615) 322-4030.

MEHARRY MEDICAL COLLEGE

Extended Continuing Education Program

Arrangements have been made with the following services and departments in the medical school to allow practicing physicians to participate in that service's activities for a period of one day to one week. This program provides an opportunity for physicians to study in depth for a specified period. The schedule of activities is individualized in response to the physician's request by the participating department. The experience includes conferences, ward rounds, audiovisual materials and contact with patients, residents, and faculty.

Fee: $75 per day or $275 per five day week of educational experience including lunch and parking at Meharry Medical College. *Credit:* AMA Category 1 of the Physician's Recognition Award, AAFP, American Academy of Family Physicians and Continuing Education Units from Meharry Medical College. *Application:* For information contact Dr. Henry A. Moses, Ph.D., Director of Continuing Education, Meharry Medical College, 1005 D.B. Todd Boulevard, Nashville, TN 37208, Tel. (615) 327-6235.

Continuing Education Schedule

Jan. 12-15	The Scientific and Clinical Basis of Biotherapy in Malignant Diseases—Franklin, Tenn. *Credit:* 28 hours AMA Category 1. *Fee:* $500.

For information contact Rosalie Avent, Biological Therapy Institute, Hospital Drive, Franklin, TN 37064, Tel. (615) 790-7535.

UNIVERSITY OF TENNESSEE

Continuing Education Schedule

Memphis

Jan. 30-Feb. 6	Medicine Review '88
Feb. 17-20	Surgical Treatment of Obesity
Feb. 27-28	Radiology
March 19-25	21st Annual Review Course for the Family Physician
April 7-9	Critical Care Symposium—Hot Springs, Ark.
June 12-19	Medical Issues in Cardiovascular, Metabolic, and Infectious Disease—Lake Buena Vista, Fla.

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Isolated Perforation of the Gallbladder Following Blunt Abdominal Trauma

WILLIAM A. WALKER, M.D. and WILLIAM L. RUSSELL, M.D.

Introduction

Injuries of the gallbladder due to blunt abdominal trauma are rare results of a direct blow to the upper abdomen or shearing forces in acceleration-deceleration injuries. Significant protection of the gallbladder is provided by its usual recessed location, partially embedded as it is in the liver, surrounded by the bony rib cage, and cushioned by adjacent viscera. This protective "shield" does not render the gallbladder immune to injury in blunt trauma, however, though the incidence of injury is difficult to accurately assess. In a large series of 5,670 patients reported by Penn[1] in 1962, including both penetrating and nonpenetrating abdominal trauma, the incidence of injury to the gallbladder was 2%. Only 8% to 10% of these injuries were due to blunt trauma. In a more recent series by Soderstrom et al[2] in 1980, of 1,449 patients sustaining blunt abdominal trauma, 31 patients were found to have trauma to the gallbladder, for an incidence of 2.1%. In contrast to earlier reports, in which wagon wheel injuries, kicks, and falls were the most common factors implicated in blunt gallbladder injuries, motor vehicular accidents are now one of the primary causes of such injuries.[2-6]

Injuries to other abdominal organs in association with gallbladder injuries are the general rule, isolated gallbladder injuries constituting only a small fraction of all gallbladder injuries proper.[2] Rupture or perforation of the gallbladder is part of a spectrum of injuries seen with blunt trauma, which also includes contusion, avulsion, and traumatic cholecystitis.

The comprehensive review of the literature regarding isolated traumatic rupture of the gallbladder by Spigos et al[3] in 1981 documented 162 cases of traumatic isolated rupture of the gallbladder with personal experience of three cases. A review of the literature since 1981 documented an additional 14 cases of isolated perforation of the gallbladder for a total of 176 cases.[2,7-15]

This paper describes a case of isolated gallbladder perforation, and reviews the clinical symptoms and treatment, with special emphasis on newer roentgenographic imaging techniques as aids in the early diagnosis of these injuries.

Case Report

After a 26-year-old intoxicated man received multiple direct blows to the upper abdomen in an altercation, he complained of diffuse upper abdominal pain without nausea or vomiting. Examination showed a slight tachycardia and mild

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From the Department of Surgery, University of Tennessee College of Medicine, Chattanooga.

Reprint requests to Department of Surgery, University of Tennessee College of Medicine, 921 E. Third St., Suite 400, Chattanooga, TN 37403 (Dr. Russell).

tenderness to palpation in the upper abdomen with voluntary guarding. Laboratory data included a serum amylase of 92 U/L (normal 22 to 85) and a WBC count of 16,700/cu mm, with 47% segmented neutrophils and no bands. A diagnosis of abdominal wall contusion was made, and the patient was discharged to jail. He returned to the emergency department four hours later complaining of increased abdominal pain localized to the upper abdomen, and accompanied by nausea and bilious vomiting. Abdominal examination showed diminished bowel sounds and marked tenderness to light palpation over the upper abdomen, with marked voluntary guarding but no rebound tenderness or rigidity. Repeat laboratory results demonstrated a rise of the WBC count to 25,000/cu mm, with 10% bands, and a serum amylase of 89 U/L. His hematocrit, liver function studies, and bilirubin were all normal. Urinalysis was positive for occult blood. X-rays of the chest and abdomen showed no free air or other pathology. After intravenous fluids and nasogastric suction were initiated, the patient was admitted to the intensive care unit for observation. Emergency CT scan of the abdomen demonstrated a normal liver, spleen, and pancreas, but there was an increased density within the gallbladder, believed most likely to represent stone formation, and an atrophic left kidney. Sonography confirmed echogenic material with the appearance of sludge in the gallbladder, most likely with stones (Fig. 1). Bilateral hydronephrosis was also noted, due to chronic bilateral ureterovesical obstruction demonstrated later by intravenous pyelography.

The patient's clinical appearance improved over the next 24 hours, with near complete resolution of his abdominal pain. His hematocrit remained stable and serum amylase remained in the normal range. Nasogastric suction was discontinued and he was started on a liquid diet. On his third hospital day he was discharged home with a follow-up appointment for further evaluation of his renal findings. At the time of discharge he was on a regular diet and ambulatory without difficulty, but still complaining of very mild, intermittent epigastric and right hypochondrial pain.

On follow-up two weeks later, the patient related having had constant right upper quadrant and right flank pain since discharge that intensified with meals and occasionally was associated with postprandial nausea and vomiting. The pain had become acutely worse several hours before his clinic visit, with near syncope. He denied urinary symptoms or change in his bowel habits, and had had no symptoms consistent with chronic cholecystitis. Examination showed an acutely distressed individual with splinting of his entire right side and inability to find a position of comfort. He was mildly tachycardic and diaphoretic. Examination of the abdomen revealed intense tenderness in the right hypochondrium and flank area, with right costovertebral tenderness and marked voluntary guarding without rebound tenderness. Laboratory data showed a WBC count of 16,600/cu mm with a moderate left shift. His hematocrit had dropped to 36%. Alkaline phosphatase was slightly elevated but the serum amylase remained normal. An emergency intravenous pyelogram to exclude an occult vascular insult to the right kidney was unchanged from the previous study two weeks earlier.

Because of the presence of gallstones found during the previous workup, a tentative diagnosis of acute cholecystitis was made and the patient was admitted to the hospital and placed on intravenous fluids, nasogastric suction, and broad spectrum antibiotics. An emergency HIDA technetium 99 scan clearly demonstrated extravasation of the radiopharmaceutical into the right paracolic gutter ten minutes after injection, consistent with perforation of the gallbladder (Fig. 2). Exploratory laparotomy revealed a large hematoma overlying the right lobe of the liver extending into the right colic gutter and the infrahepatic space surrounding the gallbladder; the omentum had walled off the right upper quadrant from the remainder of the peritoneal cavity. There was bile staining of the entire abdomen, most readily apparent in the hepatoduodenal ligament, the omentum, and the mesentery of the colon at the hepatic flexure. No active bile leak or bleeding was identified. After evacuating the hematoma, a 1-cm perforation of the gallbladder was identified along the posterior lateral border of the infundibulum proximal to the junction of the cystic duct. There was clot in the gallbladder, but no stones were found.

A cholecystectomy was performed, and a cholangiogram was normal; the infrahepatic and subdiaphragmatic spaces were drained. Histologically the cystic duct showed some signs of fibrosis and stenosis, and there was acute and chronic inflammation in the wall of the gallbladder with acute hemorrhage at the site of perforation. The patient recovered without complication and was discharged on the tenth postoperative day for outpatient follow-up. In retrospect, the density seen in the gallbladder on CT scan during his initial workup had the same attenuation coefficient of fresh blood.

Discussion

Blunt injuries to the gallbladder were classified by Penn into four major groups: contusion, laceration, avulsion, and traumatic cholecystitis.[1] Gallbladder contusions are more often concealed than revealed, and are often found only when concomitant injuries necessitate an abdominal

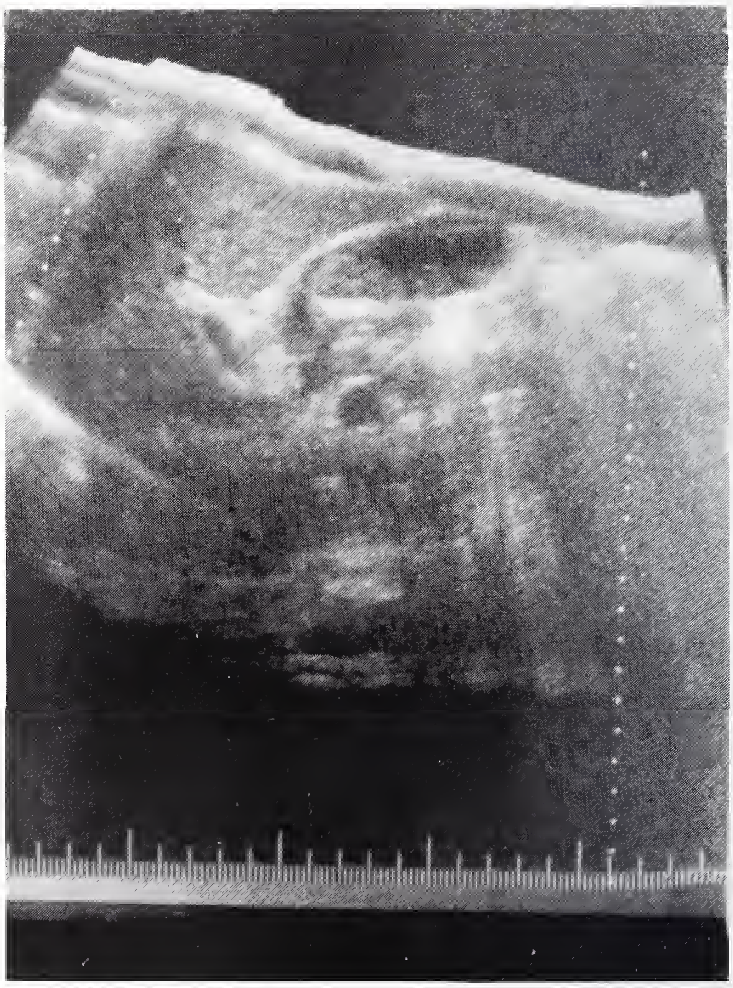

Figure 1. Sonogram of gallbladder demonstrating echogenic material with acoustic shadowing.

exploration.[2] In Soderstrom's experience, 65% of the blunt gallbladder injuries seen in a six-year period were contusions.[2] None of these were suspected preoperatively and all were associated with injuries to other abdominal organs.

The natural course of a gallbladder contusion remains unknown. Focal bruising probably resolves without significant clinical sequelae. More severe bruising may go on to heal, but no doubt can progress from echymosis to necrosis.[16] It is conceivable that patients reported to have "delayed rupture" of the gallbladder following trauma may have had an unrecognized contusion that progressed to necrosis and rupture. Delayed ruptures may also represent lacerations that are rapidly sealed off by the omentum with subsequent loss of the integrity of the omental plug and delayed symptoms of rupture. Lacerations, or "traumatic perforations/ruptures" are the most frequently reported injury of the gallbladder due to blunt trauma. Lacerations vary in size from very small ones to those encompassing large areas of the gallbladder, with a linear or stellate configuration, and most commonly involve the fundus.[3,5,16] Avulsions are the second most frequently reported injury to the gallbladder in blunt trauma, and may be either partial or complete, the latter frequently referred to as "traumatic cholecystectomy."[17] The precipitating event leading to "traumatic cholecystitis" is hemorrhage into the gallbladder lumen, either from direct, often occult, injury to the gallbladder or from hemobilia following liver injury.[18,19] Occlusion of the cystic duct by clot precipitates an attack of acute cholecystitis.

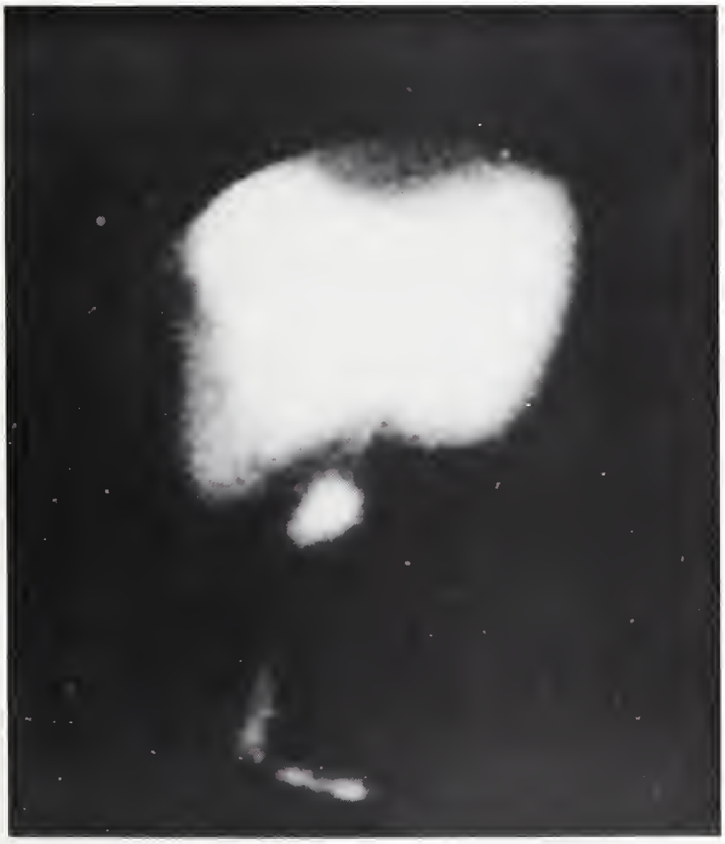

Figure 2. HIDA scan with collection of radioisotope in the right paracolic gutter and pelvis.

Historically, a wagon wheel or an assailant's foot was the most frequent offender in blunt gallbladder injuries,[1,20] but more recent reports implicate motor vehicular accidents with rapid deceleration and impalement on the steering column, or shearing forces generated by rapid acceleration-deceleration, as the leading cause of such injuries.[2,3,5]

The mechanism of gallbladder perforation is a forceful, direct blow to the upper abdomen where a direct external linear force applied to a filled gallbladder is converted to intraluminal hydraulic pressure. When the rate of generation of the hydraulic pressure exceeds the rate of dissipation of pressure by any ductal egress of bile, the tensile strength of the gallbladder wall may be exceeded, and rupture can occur.[21] The ability of the gallbladder to withstand a rise in intraluminal pressure and increased wall tension is partially conditional on the existing volume to potential volume capacity of the gallbladder; thus, the more distended the gallbladder the greater the likelihood of rupture. According to LaPlace's law, the fundus should be and is the most frequent site of perforation.[3,5,15,16,22,24]

There are three predisposing factors to rupture of the gallbladder from blunt trauma. First, the normal, thin-walled gallbladder is believed to be at greater risk than the diseased gallbladder with a fibrotic and thickened wall.[16] Secondly, the distended gallbladder in the interdigestive stage is more at risk than a contracted, nearly empty gallbladder in the digestive stage.[16] The third factor involved is ethanol ingestion. Ethanol plays a major role in blunt rupture of the gallbladder for several reasons: ethanol in the stomach increases the output of secretin and gastrin, both of which increase the flow of bile[25]; ethanol increases the tone of the sphincter of Oddi, with distension of the gallbladder and decreasing the ability of the bile ducts to serve as a vent for increasing pressures within the gallbladder[26]; ethanol causes abdominal wall relaxation, removing an important protective mechanism.[3]

Shearing forces are probably the major factor in avulsion injuries of the gallbladder.[2] The differences in mass between the liver and gallbladder could result in shearing forces between these two organs in acceleration-deceleration injuries. Soderstrom postulates that the distended gallbladder is not only more prone to rupture, but also more prone to avulsion because of its greater mass when filled with bile.[2]

The clinical manifestations of blunt gallbladder injuries vary with the severity of the injury sustained. Gallbladder contusions often produce only vague abdominal discomfort in the epigastrium and hypochondrium, often associated with mild muscle spasm and occasionally nausea. These symptoms are often worse in the postprandial state, and gradually subside with healing of the gallbladder when the contusion has been mild. With a more severe contusion the symptoms may intensify as inflammation and gangrenous changes occur, producing symptoms that are indistinguishable from those associated with acute cholecystitis.

Gallbladder lacerations or perforations produce one of two different clinical manifestations, depending on the degree of soilage of the abdominal cavity and the volume of blood lost from the gallbladder wall. With massive hemocholeperitoneum, the patient is in extremis; the flow of bile is not controlled and chemical peritonitis ensues, primarily due to the toxic effects of bile salts and the subsequent sequestration of fluid in the peritoneal cavity. Exsanguination from torn vessels in the gallbladder wall can occur in a short time.[23]

Conversely, when the laceration is small and the bile leak is minimal, or when the laceration with attendant bile and blood flow is quickly controlled by an omental "plug," a controlled choleperitoneum results, which can produce a diagnostic dilemma. The early intense pain and mild shock rapidly regress with such conservative measures as bowel rest and intravenous fluids. This quiescent period, termed the "period of illusion" by Schecter, when the bile and blood become pseudoencapsulated may last for a period of hours to weeks.[16] The patient may go on to experience continued dull pain in the upper abdomen and flank area, often made worse by meals and accompanied by nausea and vomiting. Anorexia, emaciation, hiccups, pallor, jaundice, pruritis, fecal acholia, and weight loss may accompany the persistent abdominal pain. Late complications include progressive abdominal distension from an enlarging bilious pseudocyst, or acute peritonitis and shock from disruption of the omental "wrap."

The diagnosis of rupture of the gallbladder prior to the development of such late complications is difficult. Most of these injuries are discovered at laparotomy for concomitant injuries, or rarely when the injury results in a massive hemocholeperitoneum. Isolated injuries not accompanied by peritonitis from hemocholeperitoneum are usually not diagnosed until after several hours to weeks when sequelae to these injuries develop, due mainly to the lack of any pathognomonic signs or symptoms and lack of specific chemical abnormalities in the blood.

Various diagnostic modalities have been reported to be useful for diagnosing isolated blunt rupture of the gallbladder. Paracentesis with aspiration of bile as practiced in the 19th century is totally inadequate. Bile found in the effluent during diagnostic peritoneal lavage may be helpful. While bile-stained effluent would indicate injury to the biliary system, it is not specific for rupture of the gallbladder, since it may also be seen in liver and duodenal injuries, both of which are more common than gallbladder rupture following blunt trauma. Ultrasonic examination has reportedly been useful in the diagnosis of such injuries,[3,7] with demonstration of an anechoic multiseptate mass representing a pseudocyst in the right upper quadrant, free fluid in the subhepatic space, and low-density echoes evenly distributed throughout the lumen, which suggest hemorrhage.[3,7,27] Computerized tomography may also demonstrate subhepatic fluid collections, but more importantly will demonstrate dense, intraluminal material with the same or similar attenuation coeffecient as fresh hemorrhage.[7,27] The HIDA technetium 99 scan can be diagnostic when extravasation of the radiopharmaceutical agent is seen outside the wall of the gallbladder, and the diagnosis can be inferred when the gallbladder is not seen following trauma in a patient without prior gallbladder symptoms.[7,27]

Retrospectively, the diagnosis of gallbladder injury should have been made during the first hospitalization of the patient presented in this paper; the CT scan clearly demonstrated material in the gallbladder with a similar attenuation coefficient as fresh hemorrhage. Additionally, the ultrasound studies documented low-level echogenic "sludge" in the gallbladder that should have suggested clot. On his second admission to the hospital, the HIDA scan clearly demonstrated the gallbladder and extravasation of the radiopharmaceutical agent outside the biliary tree, with collections in the subhepatic space and in the right colic gutter.

Several treatment modalities have been reported successful in the recent literature.[16,21] The treatment of choice continues to be cholecystectomy; cholecystostomy and cholecystorrhaphy should be employed only in situations where cholecystectomy cannot be performed safely.

In summary, rupture of the gallbladder due to blunt trauma is a very uncommon injury that presents a definite diagnostic dilemma. HIDA scan is a valuable diagnostic modality that can unequivocally diagnose these injuries preoperatively. The treatment of choice remains cholecystectomy.

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HELP FOR IMPAIRED PHYSICIANS

Through its Committee on Impaired Physicians, TMA helps doctors who are suffering from alcoholism and drug addiction. The thrust of the program is rehabilitative, not punitive. The Committee is composed of physicians who have special expertise in these areas, some from personal experience. Effective treatment for these illnesses is achieved most easily when the disease is detected early and family, friends, and associates are urged to avoid misguided sympathy which enables the condition to deteriorate.

HELP US TO HELP

Call the TMA Impaired Physician Program (615) 327-2711; outside Nashville call collect. Phone service available around the clock.

All-Terrain Vehicle Accidents In Robertson County, Tennessee

WALTER W. WHEELHOUSE, M.D. and NARSH D. BENSON, II, C.R.N.

An all-terrain vehicle (ATV) is defined as any motorized off-highway vehicle 50 inches or less in width, having an unladen dry weight of 600 pounds or less, traveling on three or more low pressure tires, and having a seat designed to be straddled by the operator, with handlebars for steering control.[1] Though the ATV was introduced in the United States in 1971 primarily for the farmer as a maneuverable alternative to the horse or tractor, now more than 90% of riders use the vehicles for recreational purposes.[2,3] There are an estimated 2.3 million ATVs in use in the United States, the majority of them three-wheelers.[4] The Consumer Product Safety Commission's most recent statistics show that in 1982 there were 8,600 ATV related injuries, and that in 1986 there were 86,400 ATV related injuries.[5] The Consumer Product Safety Commission also reported that between 1982 and 1986 there were 644 deaths involving ATVs, 303 (47%) of them in victims under age 16, and 135 (21%) under age 12.[5]

ATV injuries and deaths have been reported by various authors with increasing frequency over the past four to five years.[1-19] Reasons cited for the increasing rates include the following: inherent instability of the vehicle, with too high a center of gravity, a solid rear axle and an inadequate suspension system,[4-11] lack of parental supervision,[12] riding with passengers,[6,13] drinking alcohol while driving,[13,14] riding on paved roads,[15,16] driving too fast,[17] not wearing proper safety equipment (helmets, boots, and protective clothing),[7,14] inexperienced rider,[12,15,17] driver inattention,[14] riding at night,[9] and inappropriate advertising.[4]

Jesse Holman Jones Hospital is the only hospital located in Robertson County, Tennessee, a primarily rural area 30 miles north of metropolitan Nashville. In the emergency room at Jesse Holman Jones Hospital we have seen an increasing number of patients with injuries due to ATV accidents over the past three years. We undertook a review of their cases in an attempt to define the problem and to make recommendations for the reduction of ATV related injuries.

Methods

Between March 1984 and August 1986, 60 patients were seen at the Jesse Holman Jones Hospital in Springfield, Tenn., for treatment of injuries as the result of ATV accidents. Their medical records were reviewed for age of patient, sex, type of ATV (three or four wheeler), mechanism of injury, date and time of injury, type of injury, and treatment course.

Results

Sixty patients between the ages of 10 months and 69 years were treated; their average age was 17 years, and 36 (60%) were 15 years old or younger (Fig. 1). The largest single group was in the 11 to 15 year age range. The majority of pediatric-aged victims were riding without parental supervision, though the youngest victim, a ten-

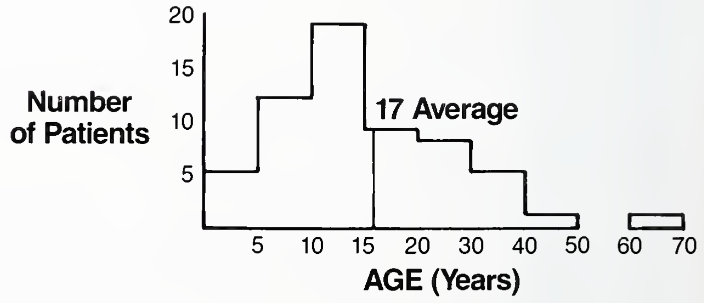

Figure 1. Age range for ATV accident victims between 10 months and 69 years of age.

From the Emergency Department, Jesse Holman Jones Hospital, Springfield, Tenn.

Presented as a scientific exhibit at the 80th annual scientific assembly of the Southern Medical Association, Atlanta, Nov. 8-11, 1986. Read before the 4th annual meeting of the Southern Orthopaedic Association, Hamilton, Bermuda, May 20-24, 1987.

Reprint requests to 514 Brown St., Suite 100-200, Springfield, TN 37172 (Dr. Wheelhouse).

month old boy, was riding as a passenger with his father.

Forty-six (76.7%) of the victims were male and 14 female. There were 53 three-wheelers (88%), and seven four-wheelers involved in the accidents. The majority of the victims were not wearing helmets or other safety equipment. Only two patients recalled wearing a helmet while riding their ATV, and one of them sustained a scalp laceration.

ATV injury classification and numbers were as follows: 25 fractures and dislocations, 23 abrasions, 22 contusions, 19 lacerations, three concussions, two sprains, one toe amputation, one spleen hemorrhage, one renal contusion, and one burn; there were no deaths. There were five hospital admissions, ranging from 2 to 15 years old, three for fractured femurs, one for toe amputation, and one for multiple injuries including rib fractures, distal right radius fracture, spleen hemorrhage, and renal contusion.

Eight patients struck barbed wire fences and sustained multiple lacerations. Thirteen patients lost control of their ATV, resulting in injuries. Three patients were injured as passengers on an ATV. Seven patients struck an inanimate object while riding their ATV, and one patient fell off a sled pulled behind a four-wheeler, resulting in a femur fracture.

ATV related injuries have been increasing in frequency in Robertson County, Tennessee, over the past three years. There were six ATV injury victims treated in 1984, 30 in 1985, and 24 through August 1986. ATV accidents occurred throughout the year, with the majority occurring during the spring and summer months; all but four occurred in the afternoon or early evening hours.

Discussion

The frequency of injuries from ATV use is increasing at an alarming rate. The majority of ATV accident victims are under 16 years old.[6,12] While there were no deaths in our series, there were five hospitalized seriously injured patients between 2 and 15 years old. Our series corroborates the high risk to adolescents, relative frequency of fractures and lacerations, and infrequent use of helmets that has come to characterize reports of three-wheeler injuries.[7] Some series suggest that four-wheelers may be less dangerous, but our study shows that four-wheeler accidents did occur in 7 (12%) of the 60 cases.[18,19] Though four-wheeled ATVs appear to be less commonly involved than three-wheeled ATVs, it is perhaps because they are relative newcomers to the marketplace.[7]

The key to prevention of ATV accidents is knowledge, safety precautions, and education. ATV use should be limited to mature, responsible, sober adults who are proficient in the use of ATVs. While helmets are strongly recommended, they alone would not have prevented the majority of injuries in our series. Other safety equipment, including gloves, sturdy footwear, and abrasion resistant clothing, is strongly advocated. The public must come to see ATVs as potentially dangerous motorized vehicles rather than toys.[7]

Conclusions

ATV accidents have been increasing over the past three years. The majority of the victims are under 16 years old, and were not wearing helmets or other safety equipment. Injuries range in severity from contusions and lacerations to fractured femurs and amputation. The majority of ATV accident victims were not proficient in the use of ATVs, and lacked the maturity, experience, and judgment to properly operate the vehicle. The key to prevention of ATV accidents is knowledge, safety precautions, and education.

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Vanderbilt Morning Report

Blastomycosis

Case Report

A 37-year-old white man was transferred to the Nashville V.A. Medical Center for evaluation of chest pain and a right upper lobe infiltrate noted on chest roentgenogram. He had had coronary artery bypass grafting six years earlier. The patient had no known tuberculosis exposure, had smoked one pack of cigarettes a day for 20 years, and had not experienced fevers, chills, night sweats, decreased appetite or weight loss.

His physical examination was unremarkable. Laboratory examination showed a WBC count of 10,800/cu mm (normal 4,800 to 10,800), with a normal differential. Hematocrit was 40% (normal 42 to 52); platelet count, SMA6, PT, PTT and urinalysis were all normal. SMA12 showed an alkaline phosphatase of 234 IU/L (normal 26 to 88) and an SGOT of 65 IU/L (normal 10 to 42). Electrocardiogram was unchanged from previous tracings. Chest roentgenogram showed a parenchymal infiltrate in the right apex (Fig. 1).

Workup for his chest pain revealed no cardiac source. A chest CT scan done to evaluate the pulmonary infiltrate demonstrated a 7-cm soft tissue mass in the apical segment of the right upper lobe; bronchoscopy revealed normal endobronchial anatomy, and transbronchial biopsies showed only inflammatory tissue. Stains for acid-fast bacilli and fungi were negative. He was treated with antituberculous medications pending culture results.

Four weeks later cultures from bronchoscopy grew *Blastomyces dermatitidis*. Isoniazid and rifampin were stopped and ketoconazole 400 mg/day was begun.

Discussion

Blastomyces dermatitidis is a thermic dimorphic fungus. Infection is acquired by inhalation. Nonpulmonary involvement, including skin lesions, is the result of lymphatic and hematogenous dissemination from a primary pulmonary focus.[1] Cases in this country are predominantly in the southeast and south central United States and in the Great Lakes area. It tends to occur in young and middle aged men; exposure to soil and wooded areas may increase the risk of infection.[1,2]

Blastomycosis ranges from an accidentally discovered asymptomatic self-limited pulmonary process to a progressive and fatal disseminated illness. It may produce an acute pneumonic syndrome that progresses to a chronic pulmonary infection with or without disseminated disease. In the latter case the illness pursues a low grade yet progessive course.[1,2]

In 20% to 90% of cases of blastomycosis there is evidence of multiorgan involvement, the skin being the most common site of dissemination. There is bone involvement in 25% of patients, and the male genital tract may also be involved.[1,2]

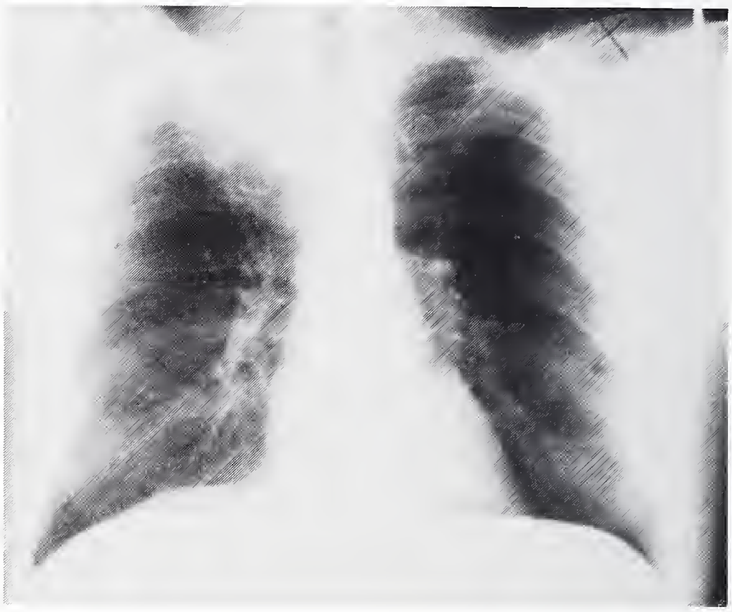

Figure 1. Chest roentgenogram showing a right upper lobe infiltrate.

Establishing the diagnosis of blastomycosis requires demonstration of the organism in biopsy specimens or culturing it from infected tissue or body fluids. Noncaseating granulomas may be present in tissue specimens[1,2]; specific skin tests and serologies are of limited use. Chest roentgenogram findings are extremely variable. There may be hilar adenopathy and pleural thickening, as well as homogenous pneumonic infiltrates with segmental or lobar distributions, patchy fibronodular or diffuse nodular infiltrates, fibrotic changes, and cavities.[3,4]

In patients with disseminated and severe illness, the recommended therapy is systemic amphotericin B, but for acute pulmonary infection recommendations are variable, some authors arguing that patients should be observed without therapy for two to three weeks and then treated only if symptoms do not resolve. Others argue that ketoconazole should be given for six months, in part to limit dissemination.[5,6]

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A Case of Alcoholic Ketoacidosis

Case Report

A 68-year-old white woman transferred to Vanderbilt Hospital for severe metabolic acidosis had a history of heavy alcohol consumption of up to one quart of scotch per day for at least several years. Her appetite was generally poor, and her daily activities were minimal. She was in her usual state of health until two days prior to transfer when she developed nausea and vomiting without abdominal pain or other symptoms. She stopped drinking, but when her vomiting persisted she was taken to her local hospital where she was noted to be lethargic and pale. Arterial blood gases (ABGs) showed a pH of 6.97 (normal 7.35 to 7.45), Po$_2$ 122 mm Hg (normal 80 to 100), Pco$_2$ 14 mm Hg (normal 35 to 45), and calculated bicarbonate 4 mEq/L (normal 24 to 30). Her serum sodium was 131 mEq/L (normal 135 to 145), potassium 3.7 mEq/L (normal 3.5 to 4.5), chloride 86 mEq/L (normal 95 to 105) and glucose 219 mg/dl (normal 70 to 110). She was given sodium bicarbonate, fluids, and glucose intravenously, and transferred to Vanderbilt.

Physical examination showed a thin, chronically ill appearing white woman with moderate tachypnea and a temperature of 100°F rectally, but her other vital signs were normal. Physical examination showed clear lungs and normal heart sounds, her abdomen was mildly tender, and her liver was palpable 2 cm below the right costal margin. There was no splenomegaly and bowel sounds were normal. Stool was heme-negative. Laboratory examination showed a serum sodium 143 mEq/L, potassium 4.3 mEq/L, chloride 101 mEq/L, bicarbonate 9 mmole/L (normal 23 to 30) and glucose 171 mg/dl, and ABGs showed pH 7.21, Pco$_2$ 15 mm Hg, and O$_2$ 146 on 2 liters O$_2$. Her WBC count was 14,400/cu mm (normal 5,000 to 10,000) with 88% segmented neutrophils, 5% lymphocytes, and 7% monocytes. Hematocrit was 37% (normal 37 to 41) with an MCV of 112 cuμ (normal 80 to 100) and platelet count was 154,000/cu mm (normal 150,000 to 350,000). PT, PTT, liver function tests, and serum amylase were normal. Urinalysis showed 4+ acetone and no crystals. Drug and toxin screen showed no evidence of salicylates, ethanol, methanol, ethylene glycol or other substances. Serum ketones were 1:8 (normal—undetectable), and serum lactate was mildly elevated. KUB and chest films were normal, and blood and urine cultures were negative.

The patient was treated with fluids and glucose and her acidosis resolved entirely within 24 hours. Alcoholic rehabilitation was recommended but the patient refused.

Discussion

Alcoholic ketoacidosis was first reported in the medical literature over 40 years ago and has since been well described.[1] It most frequently occurs in severe chronic alcoholics who develop abdominal problems such as gastritis and pancreatitis, leading to vomiting and an inability to consume food or alcohol. In fact, most patients who develop alcoholic ketoacidosis have not drunk alcohol for one to three days.[1] Physical examination of these patients is usually unremarkable except for tachypnea and ketone odor on the breath. Abdominal pain is not uncommon, but intra-abdominal catastrophe is rare.

Laboratory findings usually reveal a metabolic acidosis with an elevated anion gap, although an alkalosis or mixed acid base disturbance may be present due to concurrent tachypnea or vomiting[2]; serum glucose values are variable, but serum ketones are markedly elevated with an increase in the beta hydroxybutyrate/acetoacetate ratio, while serum lactate may be slightly increased.[1]

The pathophysiology underlying alcoholic ketoacidosis is only partially understood. Most patients are starved for several days before they come to a physician. Alcohol suppresses ketogenesis in this situation and its withdrawal, coupled with low insulin and high glucagon levels, leads to release of fatty acid stores, flooding of the liver with these compounds, and subsequent ketone body production.[1]

The treatment of alcoholic ketoacidosis is not difficult; replacement of fluid and carbohydrate stores usually corrects the metabolic imbalance within 24 to 30 hours. Insulin is not necessary. Any other electrolyte disturbances should be corrected.

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TENNESSEE MEDICAL ASSOCIATION

153RD ANNUAL MEETING

April 13-16, 1988

Peabody Hotel, Memphis

Radiology Case of the Month

GARY H. MORGAN, M.D. and STEPHEN L. GAMMILL, M.D.

Case Report

A 26-year-old white gravida 1, para 0, with an uneventful prenatal history went at 29 weeks' gestation to a local emergency room complaining of severe right upper quadrant pain radiating to her back and right shoulder for three hours.

She had nausea and vomiting after eating her dinner, but denied abdominal trauma, genitourinary symptoms, or change in stool. Physical examination revealed a positive Murphy's sign, a palpable uterine fundus, and normal bowel sounds, but no evidence of hepatosplenomegaly, jaundice, or peripheral edema. She was afebrile, and had a blood pressure of 190/120 mm Hg and heart rate of 70/min. Hemoglobin and hematocrit were 13.9 gm/dl and 41.5% respectively. WBC count was 14,200/cu mm, with 77% segmented neutrophils and 19% lymphocytes; platelet count was 222,000/cu mm. Urinalysis revealed 2+ albumin, electrolytes were normal, serum amylase 90 IU/L, alkaline phosphatase 171 IU/L, LDH 365 IU/L, and SGOT 120 IU/L. Ultrasonography of the abdomen was negative.

The patient was admitted to the labor and delivery suite, where uterine sonography demonstrated a single viable intrauterine fetus in cephalic presentation at 28 weeks ± 2 weeks gestational age; there was no fetal movement except for normal cardiac activity. Initial laboratory data included hematocrit 38.3%, platelets 121,000/cu mm, SGOT 58 IU/L, LDH 201 IU/L, SGPT 69 IU/L, GGT 19 IU/L, and alkaline phosphatase 135 IU/L. Persistently elevated blood pressure and decreased urine output prompted emergency cesarean section, with successful delivery of a 1,000-gm infant with Apgar scores of 5 and 8. Exploration of the right upper quadrant demonstrated a normal liver edge.

Three hours after cesarean section the patient developed severe right chest pain and hypotension (62/45 mm Hg). Arterial blood gases and EKG were within normal limits. Hematocrit and platelets were 28.1% and 73,000/cu mm respectively. The patient was successfully treated with intravenous fluid and blood products.

On the first postoperative day the patient continued to complain of upper right quadrant pain. Her blood pressure was again elevated to 169/110 mm Hg, and repeat blood work showed hematocrit 30.9%, platelets 86,000/cu mm, and evidence of hemolysis on peripheral blood smear; partial thromboplastin time was 25.8 seconds, fibrin degradation products 16 to 32 mg/dl, SGOT 1,138 IU/L, LDH 2,016 IU/L, SGPT 621 IU/L, GGT 26 IU/L, alkaline phosphatase 112 IU/L, total bilirubin 3.1 mg/dl, and direct bilirubin 1 mg/dl.

Real-time sonography of the upper abdomen and computerized axial tomography (CT) with oral contrast and with and without intravenous contrast were performed to evaluate her right upper quadrant pain and elevated liver function test. Please examine Figs. 1 and 2, and then formulate your diagnosis.

Discussion

Severe preeclampsia can be diagnosed when one or more of the following develop: systolic blood pressure of 160 mm Hg or more, or diastolic pressure of 110 mm Hg or more on two readings six hours apart; proteinuria of 5 gm or more per 24-hour urine collection; cerebral or

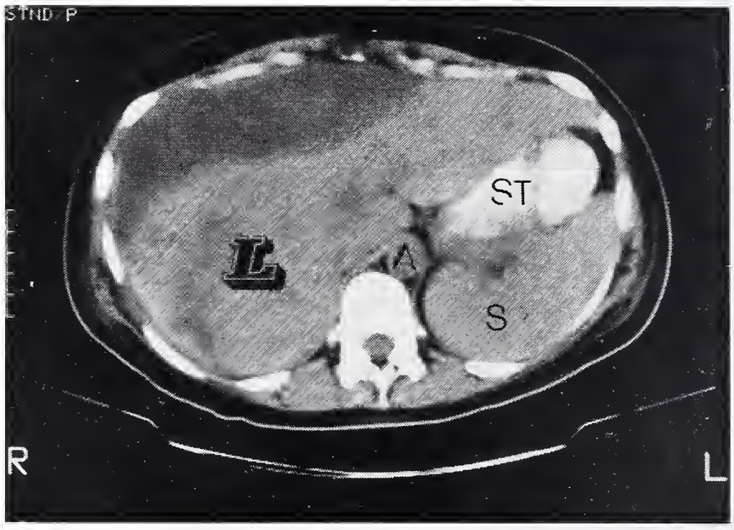

Figure 1. Lenticular fluid collection of mixed attenuation along the anterior aspect of the right lobe of the liver is compressing the liver and displacing it medially (L = liver; ST = stomach; S = spleen; A = aorta).

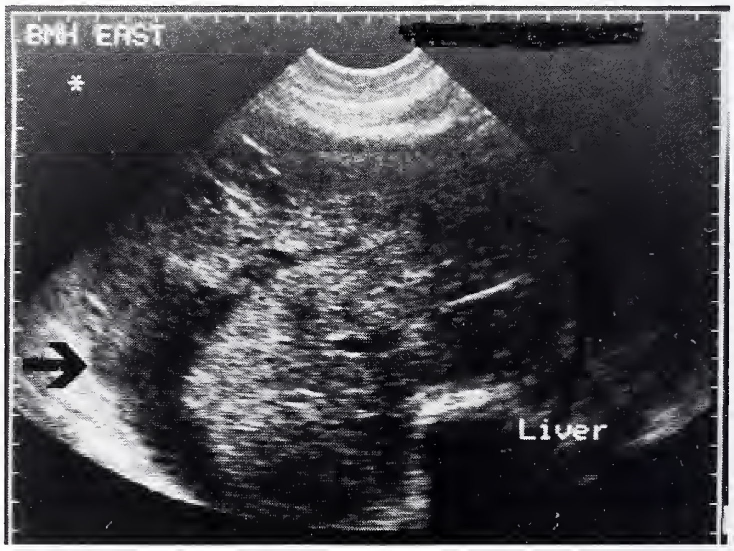

Figure 2. Fluid collection of mixed echogenicity is present along the right lobe of the liver (arrow).

From the Department of Radiology, Baptist Memorial Hospital, Memphis.

visual symptoms; pulmonary edema or cyanosis; or epigastric pain. Approximately 5% of pregnancies are complicated by preeclampsia; it tends to occur more frequently in primigravidas, particularly very young or older women, than in multiparas. The signs appear usually after the 20th week of pregnancy (particularly the third trimester) except in the rare instances when it is associated with a hydatiform mole.[1]

Weinstein reported an additional criterion that should be added to the above list of findings in severe preeclampsia. In 1982 he reported the HELLP syndrome (hemolysis, elevated liver function test, and low platelet counts) in a series of 29 patients with severe preeclampsia. It may occur when the usual findings of severe preeclampsia are absent.[2]

The liver is not primarily involved in early preeclampsia, but it is a target organ that is damaged as the disease progresses. Laboratory evaluation typically reflects hepatocellular dysfunction, with the most pronounced elevations developing in the serum transaminases. The SGOT is elevated in 50% of women with severe preeclampsia. Most people believe the basic anatomic alteration in the liver to be fibrin thrombi in the sinusoids and periportal hemorrhage in the peripheral subcapsular area. The obstruction of the blood flow in the sinusoids by intravascular fibrin deposition is a probable cause of liver distension, which results in right upper quadrant or epigastric pain. The hemorrhage is variable; it may be diffuse, or it may produce extensive destruction of the organ and/or subcapsular hemorrhage. If intrahepatic pressure exceeds the ability of Glisson's capsule to distend, the liver ruptures, with hemorrhage into the peritoneal cavity.[2-4]

While rupture of the liver occurs most commonly with trauma in the nonpregnant, 80% of women with spontaneous hepatic hemorrhage in pregnancy are toxemic. The clinical features include sudden epigastric or right upper quadrant pain with radiation to the back or shoulder, and nausea and vomiting. Because the principal signs of toxemia go unnoticed by patients, those with poor prenatal care may have advanced disease and hepatic manifestations as their presenting complaint. Although toxemia is more common in primiparas, two-thirds of the subcapsular hemorrhages occur in multiparas.[5,6]

The usual CT appearance of subcapsular hematomas is a crescentric or lenticular, well-marginated, low-density fluid collection just beneath the hepatic capsule. Intrahepatic hematomas produce round to oval collections within the hepatic parenchyma. Hepatic lacerations appear as irregular clefts or masses within the liver parenchyma, and often extend to the periphery of the liver. As elsewhere in the body, recent hemorrhage and clot have a higher density than older hematomas that have matured.[7] Sonography of a subcapsular hematoma shows a sonolucent collection within the parenchyma or beneath the capsule if it is recent; later, as the hematoma organizes, the collection becomes more echogenic, but the collection may become sonolucent again as a hygroma is formed.[8] There is a tendency for the hemorrhage to be most marked in or limited to the anterior and superior aspects of the right hepatic lobe, with rupture of the capsule at the inferior margin, despite eclamptic hemorrhages being abundant beneath the capsule in all surfaces.[3,9] Manas concluded that once hepatic hemorrhage was suspected, abdominal CT was the most helpful diagnostic tool. CT scans in all seven patients in his series showed evidence of subcapsular hematoma, intrahepatic hemorrhage, or both. CT also provided information concerning presence or absence of peritoneal fluid, suggesting oozings or rupture of Glisson's capsule.[10]

DIAGNOSIS: Subcapsular hepatic hematoma without rupture, due to severe preeclampsia and the HELLP syndrome.

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Loss Prevention Case of the Month

Fundamentals Fundamental

J. KELLEY AVERY, M.D.

Case Report

A 24-year-old woman was admitted at term to a rural hospital after an uneventful prenatal course; she was having irregular, but painful, uterine contractions. On admission at 3:30 PM examination by the labor and delivery (L&D) nurse revealed a 3-cm dilation, 75% effacement, and the presenting part at 0 station. When the attending family physician was notified, he ordered the routine enema and preparation, which were accomplished.

The patient continued to have some irregular contractions, but she was ambulatory and slept at intervals until active labor began at about 5:30 AM, approximately 14 hours after admission. Vital signs reported at half hour intervals continued to be normal; fetal heart tones (FHTs) reported at intervals of 30 minutes to one hour ranged between 130 and 150/min.

The bag of waters (BOW) spontaneously ruptured at 12 noon, some 21 hours after admission, and the attending family physician saw his patient in the L&D suite for the first time. His examination revealed normal vital signs, with contractions occurring every 5 minutes with a duration of 40 to 60 seconds; FHTs continued to range between 130 and 150/min.

There was a gradual increase in the intensity of contractions for the next few hours, and at about 5 PM the cervix was completely dilated and the head was at a +2 station.

The patient was brought to the delivery room at 6:00 PM. At 6:10 the FHTs were discovered to be absent and the attending physician was summoned stat; the patient was delivered promptly of a male infant with Apgars of 2 and 5. Spontaneous respiration was delayed for 20 minutes and the infant developed seizures at 12 hours of age; at six weeks the infant had ample physical evidence of a severe neurological deficit. Within eight months of delivery, a lawsuit was filed against the attending family physician for negligence in his management of the patient's labor and the hospital for negligence in not providing external fetal monitoring (EFM) for the obstetrical patients.

Examination of the medical record revealed that the last recorded FHT was at 5:05 PM, when the rate was documented as being 144/min. As the legal positions of the defendant hospital and the physician were fully explored, it became apparent that an early settlement on the part of both was the wisest course to follow.

Loss Prevention Comments

The indefensible position of the hospital was not due to its failure to provide external fetal monitors, since ample proof was available that EFM is not the standard of care for all patients in labor, and the current guidelines of the American College of Obstetrics and Gynecology (ACOG) do not mandate universal EFM. The lack of regular monitoring of FHT by competent observers, however, cannot be defended. This failure militated against defending both the hospital and the physician. The absence of recorded FHTs at reasonable intervals (15 to 30 minutes) indicated that neither the nursing staff nor the physician was acting within an acceptable standard of care.

It was also decided that attendance by the family physician only once during the patient's 24 hours in the L&D area of the hospital indicated an indefensible attitude of indifference, and would never have been understood by a jury.

In most medical malpractice lawsuits, it is neither the presence or absence of sophisticated technology nor its function or malfunction that causes the problem. It is the behavior of the treatment team that is critical to preventing, or prevailing in the event of, a bad result.

Dr. Avery is chairman of the Loss Prevention Committee, State Volunteer Mutual Insurance Company, Brentwood, and medical director for Ambulatory/Outpatient Services, St. Thomas Hospital, Nashville.

president's page

JAMES T. GALYON

Untapped Resources

It has occurred to me that we have some resources that are not being fully utilized. During these hectic days, medicine is making huge strides in improving the health of the nation with innovative treatments unheard of a few years ago, and yet at the same time we are rapidly losing ground on the social and economic fields. We have criticism from the press because a few would not jeopardize their lives to treat AIDS patients. We are castigated by government, business, and the insurance industry because the cost of health care continually goes up. The politicians locally and nationally seek to balance budgets by failing to fund those programs that would compensate physicians for caring for the sick and injured. Where do we turn for allies?

The first source of assistance is as near as our spouses. The TMA Auxiliary is doing a marvelous job for us. They raised more funds for AMA-ERF than any other state, a truly monumental task considering the potential of some of our more wealthy states. They also put together Teen Health Workshops to alert our young people to the hazards of drugs, alcohol, teen pregnancies, and other social problems. They "manned" telephone banks to lobby our political leaders in our interests. Still, many of our members do not encourage their spouses to belong to the Auxiliary. Think how much help we could have if we all encouraged our spouses to join the Auxiliary and be active.

Our second underutilized asset is our IMPACT-AMPAC organizations. Tennessee ranks 38th among the states in participation in these PACs. Only 15% of our members belong and contribute. In spite of this meager funding, they do a yeoman job in pursuing our interests. The PACs had a considerable part in preventing RAP physicians from coming under DRGs. With their help, we also defeated mandatory assignment three times in the House Ways and Means Committee this fall. The PACs have represented our views on professional liability legislation, Medicare reform, campaign financing, etc. While lobbying is essential, without the more tangible efforts of the PACs to support our elected officials, we could not do the job. King Canute could not hold back the tide with words and neither can we. We need money and work to stem the political and social tides.

The third source of help is to be found within ourselves. For years many physicians have held themselves aloof from politics, saying they would practice good medicine and leave the politics up to those more interested or adept. No longer can we afford that kind of political isolationism. We can see that someone is going to manage the system in which we practice. We can become personally involved with our own destiny or leave it up to others with entirely different goals. Someone will make these decisions; the choice is up to us.

All of these available resources require some commitment from us—commitment of time, money and support. We can continue to ignore these means, and trust to others to preserve our way of practice, or we can do everything possible to win this struggle. You can be sure the resources organized against us are great. We must utilize all of our assets to prevail.

James T. Galyon, M.D

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DECEMBER, 1987

editorials

Onward and—Upwards?

Apropos of what I don't remember, but the other evening in a casual conversation a friend of mine, a well-preserved man several years the far side of 70, remarked that he has always regretted that he is not an educated man. He is; what he meant was he never went to college. They are not the same thing.

My father, who if he were alive today would be something over a hundred years old, decided that rather than go to college, he would make his mark in the business world. He did; by the time he was 45 he owned his own thriving business, but he never let my brother and me forget the value of an education, because like my friend he regretted his lack of what he thought was one, particularly because he passed up the opportunity. I considered him then, and still do, one of the best educated men I ever knew outside of a university faculty. He acquired an extensive library, and read even more widely than that. He was a mathematical wizard, a talent he passed on to my brother, but not to me.

There is a vast misunderstanding, I think, about what an education is, and even among the knowledgeable there is disagreement. It has become abundantly clear that having been to college, or even holding a college degree, does not guarantee an education; a degree simply guarantees that one has a degree. There is such preoccupation with that these days that the education is overlooked; too many people are in college who should not be there, not necessarily for want of intellect. Some are simply not bright enough, of course, and some are not interested enough. Far more, though, are simply ill prepared. The problem begins at various stages, for some pre-school. Some have slovenly minds, but what is sadder is that many who do not, have had teachers with slovenly minds; many of their pupils never recover, even with a college degree.

Much has been written lately about the appalling ignorance of our young, particularly regarding history—not just ancient history, either, but our own—and literature. In a test given to approximately 8,000 high school juniors nationwide, although 80% knew of George Washington, for instance, and had a rough idea of when he lived, less than half could place Thomas Jefferson's presidency between 1800 and 1820, and only slighty more placed Franklin Roosevelt's in the 1930s. Two thirds had not even the foggiest notion as to when the Civil War was fought, and 40% of them thought World War I occurred before 1900. Some schools did far better than others in preparing their students, of course, which means that the lack of learning of the worst was truly appalling. These are not stupid students, but a cross section of our youth; they are not dumb, but ignorant. As they enter society to take their place as citizens and parents, though, it will be hard to tell the difference. It will be even harder when it comes to evaluating their offspring.

The education community cannot make up its mind about what it is it wishes to accomplish in fulfilling its obligation to our young. By law it must work with often recalcitrant teenagers to age 18, some of whom do little but disrupt the process and torment their instructors. Too, their advancement is often far beyond their accomplishments; sooner or later that truth will out, but often it is only much later. Despite this, there is entrenched wisdom that everyone is educable, and that in a democracy one must operate on this assumption, any other being elitist. That only inhibits the learning process in the more able and interested, and puts the United States at a serious and progressive disadvantage in competing with such totalitarian cultures as the ones in the Soviet Union and China. Despite our own misgivings, no such aberrations of fancy bother the European democracies, who do not find any such misperceived lack of egalitarianism undemocratic.

It has somehow gotten into the heads of certain of our leadership that "all men are created equal" means that all men (and women, of course—and girls and boys) have the same potential, even though when they go to the racetrack those same ninnies are very choosey about which bit of horse flesh is allowed to affect their own fortunes. Not every nag is given an opportunity to run at Churchill Downs. Though most people are smarter than most horses, not every one of either is suited to the fast track. Since God loves every individual equally, we are told to do the same. Mr. Jefferson meant that for that reason it should be that any American might one day become president; for various reasons, one of them having to do with mental capacity, not every one of them can. It is a subtle distinction, but a real one—one that appears lost on many of our leaders. Though he may have to struggle for it, anyone can also get an education if he is of a mind to, but some need all the help they can get. Such help is often meagre, partly because so much of what talent there is in the teaching profession is being wasted trying to push spaghetti.

In speaking about the previously mentioned test given our high school juniors, two of our country's foremost educators, who were being interviewed on *Firing Line,* instead of being distressed about the 40%, were pleased that 60% of the students knew that World War I was fought in this century; after all, they said, it happened a long time ago—much longer ago than when we (they said) were teenagers. You will not be surprised to learn, I am certain, that Mr. Buckley was appalled, nor, I hope, that I was too.

With such leaders in education it is no wonder we is in the fix we is in—which, I must add, they do not find such a bad one. God help us!

J.B.T.

God Jul—Mele Kalīkimaka—Buon Natale

> Render unto Caesar the things that are Caesar's
> And unto God the things that are God's.
>
> Matthew 20:21

During the past few days the stock market has been doing reprehensible things; some individuals riding high on the crest of the bull market have found their net worth halved. Though this does not portend financial ruin for very many of them, it has been a sobering experience—a new one for many a youngster. Gloom is settling over the mercantile community as it envisions a dull Christmas season regardless of the market's ultimate destination, with parsimony replacing profligacy. Despite those dire predictions, that does not guarantee a dull Christmas.

On Christmas Eve in 1818 the dilapidated organ in the little church of a small Austrian hamlet lay in abject disrepair—scarcely a harbinger of *Fröhliche Weinachten.* Even as he wondered what he would do for Christmas music, the village priest, Father Joseph Mohr, sat down and wrote a poem that proclaimed in a simple way the message of that first Christmas. Sung that night to the guitar accompaniment composed for the occasion by organist Franz Gruber, "Stille Nacht, Heilige Nacht" began its reign as the best known and best loved of all the Christmas carols—

Until Irving Berlin's "White Christmas" came along. Now I understand, of course, that strictly speaking, "White Christmas" is not a Christmas carol. A carol is by definition a song of praise—or joy. On the other hand, I think one need not bend the connotation too far to include "White Christmas" under that rubric. But no matter.

As celebrated since its inception some four centuries after the estimated birthdate of Jesus of Nazareth, Christmas has had one foot in the world and one foot in Heaven, with the greater weight on one or the other foot depending upon the times and the circumstances. The day belongs to God, to be sure, but it also belongs to Caesar—for better or worse.

In his wise answer as to whether or not the Roman taxes should be paid, Jesus rendered no moral judgment against the Roman statute, as His oppressors had wished He might. His injunction to render unto Caesar was without rancor. As a man who enjoyed a celebration, He would doubtless approve the sentiments expressed in the song "White Christmas," which describes an idealized celebration of it, one that many of its singers could have known only through such movies as *Holiday Inn*, in which the song appeared. Though such a Christmas is a part of Caesar's show, it is not inappropriate to Jesus' birthday party, either—

Provided all things are kept in perspective, and that it is not the whole cloth. For Christian or Jew, the season is a time for God, and through Him for the family. The temptation is always there to celebrate the pagan Saturnalia, the progenitor of the feast of Christmas. The Saturnalia was a revelry, a time of debauchery and getting. Whether the Feast of Lights or of Christ's Mass, it is to be a time of worship and giving. Even a religious feast (I hestitate to say "Holy Day," but it also) can be debauched, as the Saturnalia was. The Saturnalia started out as a day of gift-giving and peacemaking; it ended up a week-long revel. So can Christmas, and it has on occasion.

Christmas can be as bad or as good, as significant or as meaningless, as the individual celebrant or non-celebrant wishes to make it. It is a man-made celebration, since neither Jesus nor the early church made anything at all of it. The church has consecrated the day to God; the World has rendered it unto Caesar.

However you choose to keep it, or even if, like Scrooge, you choose not to keep it at all, may the day be a happy one for you and yours; if you leave God out, though, you will be the poorer for it.

J.B.T.

Corrigendum—The Long Way 'Round

On being confronted with an error in one of his editorials, a prominent editor, unless memory fails me the late great Franz Ingelfinger of the *New England Journal of Medicine*, is said to have retorted that he knew it; he only wanted to see if anyone was watching. The story may be apocryphal, but that's one approach.

Another is to blame it on someone else, and still another is to make excuses. Since I can find no one else to blame, that last is the route I'm going to take. I just picked up the October issue of the *Journal* and turned to my editorials, and couldn't believe what I saw—first thing, right off. There, gazing up at me in all their black and white splendor, were the initials *FDA*. I had written it, and had read over it at least a dozen times, including in galley, but obviously had never actually seen it. The error is in the next to last sentence in the editorial entitled "On Playing Drugstore." The initials should have been *FTC*. My apologies to the FDA—I think.

The reason I say "I think" is that though I didn't mean to say that, it just might fit, and maybe if I had kept quiet, no one would have noticed. One has to wonder how often things get screwed up in those acronymic bastions, Washington and the military (where it all started), simply because someone meant one and said another.

One of the things that livens up TV (or radio) shows is personal feuds, such as the famous one between Jack Benny and Fred Allen. Practically all are contrived, simply for the sake of viewer (theirs was listener) interest. On Nashville's *early* (5:30 to 7:00 AM) live show a new one has developed between host Ralph Emery and Channel 4's newest—and youngest—sports reporter Robin Roberts. Whenever Ralph has made light of Robin's youth by such nicknames as "cub," for example, she refers to him as "Pops," or the "old man." The other day, when Ralph was groping for a word, Robin said, "Well, you know what they say: The mind is the first thing to go." To which Ralph replied, "That's what you think—because you're young."

For the sake of the nation one hopes there is not mass confusion in Washington over all the initials of agencies they have to cope with every day. On the other hand, for one's own sake one must sometimes hope there is.

J.B.T.

in memoriam

George E. Cox, age 66. Died October 15, 1987. Graduate of Medical College of Virginia. Member of Chattanooga-Hamilton County Medical Society.

H. James Crecraft, age 67. Died September 22, 1987. Graduate of University of Maryland School of Medicine. Member of Nashville Academy of Medicine.

Ebert M. Mohler, Jr., age 68. Died September 16, 1987. Graduate of University of Tennessee College of Medicine. Member of Sullivan County Medical Society.

new members

The *Journal* takes this opportunity to welcome these new members to the Tennessee Medical Association.

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY
Bruce D. Pancake, M.D., Chattanooga

KNOXVILLE ACADEMY OF MEDICINE
Richard H. Mays, M.D., Knoxville
Anthony W. Morton, M.D., Knoxville

LAKEWAY MEDICAL SOCIETY
Kim A. D. Collinson, M.D., Morristown

MONTGOMERY COUNTY MEDICAL SOCIETY
Danny W. Futrell, M.D., Clarksville

NASHVILLE ACADEMY OF MEDICINE
William Loomis Furlow, M.D., Nashville
L. Dean Knoll, M.D., Nashville

TMA Members Receive AMA Physician's Recognition Award

Nineteen TMA members qualified for the AMA Physician's Recognition Award during September 1987.

To qualify for the PRA, a minimum of 150 hours of continuing medical education must be earned over a three-year period; 60 of these hours must be Category 1.

This list does not include members who reside in other states. Names of additional PRA recipients will be published as they are received from AMA.

Edward P. Caldwell, M.D., Memphis
Michael S. Cardwell, M.D., Brentwood
Thomas H. Curtis, M.D., Chattanooga
Robert Diez D'Aux, M.D., Greeneville
Harry Friedman, M.D., Memphis
Conrad L. Grabeel, M.D., Knoxville
Joel T. Hargrove, M.D., Columbia
George J. Heard, M.D., Columbia
Richard A. Krause, M.D., Chattanooga
Frank London, M.D., Knoxville
Herbert J. Michals, M.D., Kingsport
Augustus L. Middleton, M.D., Jackson
Frances K. Patterson, M.D., Knoxville
Michael S. Pippin, M.D., Nashville
James A. Pitcock, M.D., Memphis
Richard J. Sanders, M.D., Madison
Robert A. Valosik, M.D., Nashville
Ann O. Wehr, M.D., Nashville
Robert H. Williams, M.D., Chattanooga

personal news

J. Kelley Avery, M.D., Nashville, has been appointed by the Governor to serve on the Licensing Board for Health Care Facilities. Dr. Avery, a past president of TMA and formerly medical director for SVMIC, is the medical director for ambulatory-outpatient services at St. Thomas Hospital, where he also serves as the medical director for continuing education.

Danny W. Futrell, M.D., Clarksville, has been certified as a Diplomate of the American Board of Pediatrics.

John H. Grant Jr., TMA Director of Education and Meeting Services, has earned the Certified Association Executive (CAE) designation of the American Society of Association Executives (ASAE).

Frank Pennington, M.D., Jackson, has been inducted as a Fellow of the American Academy of Facial Plastic and Reconstructive Surgery.

George A. Zirkle, M.D., Knoxville, has been named Pediatrician of the Year by the Tennessee Pediatric Society. Dr. Zirkle, a past president of TMA, has been in active pediatric practice for 37 years, and has contributed greatly to his profession on the local, state, and national levels.

The following TMA members have been certified as Diplomates of the American Board of Family Practice: *Kelly Ballard, M.D.*, Jackson, and *Claude Bridges, M.D.*, Dyer.

The following TMA members have been honored by the American Academy of Family Physicians in recognition of their long periods of continued membership in the Academy: *Sam Luton, M.D.*, Clarksville, 25 years; and *Marvin M. Nathan, M.D.*, Chattanooga, 35 years.

announcements

CALENDAR OF MEETINGS

NATIONAL

Jan. 10-13	Inter-American Society for Chemotherapy—Clearwater, Fla.
Jan. 18-24	Southern Clinical Neurological Society—Fort Myers, Fla.
Feb. 1-3	Southeastern Surgical Congress—Lake Buena Vista, Fla.
Feb. 2	American Association of Psychiatric Services for Children—San Antonio, Tex.
Feb. 3-5	Southern Society for Pediatric Research—Hyatt Regency Hotel, New Orleans
Feb. 7	American Orthopaedic Foot and Ankle Society—Atlanta
Feb. 11-13	Society of University Surgeons—San Antonio, Tex.
Feb. 15-19	Society of Toxicology—Dallas
Feb. 17-21	American College of Psychiatrists—Tucson
Feb. 18-21	American College of Cryosurgery—New Orleans
Feb. 27-March 5	North American Trauma Association—Snowmass Village, Colo.
Feb. 28-March 2	Society of Air Force Physicians—La Mansion Del Rio on the Riverwalk, San Antonio, Tex.
Feb. 28-March 4	United States and Canadian Academy of Pathology—Washington Hilton, Washington, D.C.

Charlie Cato Added to TMA Staff

TMA Executive Director L. Hadley Williams has announced the appointment of Charles W. Cato, II as TMA's Director of Legislative and Governmental Affairs. Mr. Cato joins the TMA having formerly served as corporate counsel for the Tennessee Hospital Association. Prior to his THA position, he held various positions on staff in the Tennessee General Assembly, he served as administrative assistant to the Senate Minority Leader, and he was an employee in the office of the Chief Clerk of the Senate.

In addition to his broad legislative experience, Charlie was engaged in the general practice of law for three years after receiving his doctor of jurisprudence degree from Vanderbilt University in 1979 and admission to the bar in 1980.

Cato graduated from Southwestern in Memphis with a bachelor of science degree in economics in 1976. In addition to his legal training, he has passed the CPA examination and anticipates being licensed in Tennessee upon satisfaction of the experience requirement.

Charlie is married to the former Merry Lynn Doster of Lookout Mountain, and is the father of one son, Chase, age 6 months.

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Highlights of the TMA Board of Trustees Meeting

October 11, 1987

The following is a summary of the major actions taken by the Board of Trustees of the Tennessee Medical Association at its regular fourth quarter meeting at TMA Headquarters in Nashville on Oct. 11, 1987.

	THE BOARD:
Establishment of a Trauma System in Tennessee	Voted to go on record as supporting efforts to develop a trauma system throughout the state and the Emergency Medical Services Committee's recommendation that such a system follow the guidelines established by the American College of Surgeons.
Committee on Mental Health	Voted to draft legislation to change the Tennessee statute regarding commitment of incompetent patients to include the seriously ill psychiatric patient. The Board also agreed that the Mental Health Committee should draft such legislation if the Commissioner of Mental Health and Mental Retardation does not seek to repeal existing law by July 1, 1990.
Conference on Drug Prescribing Environment	Voted to request the Tennessee Academy of Family Physicians to consider sponsoring a conference on the Drug Prescribing Environment during their 1988 fall meeting, along with other medical specialty societies as appropriate.
Rural Health Committee	Received a report from the Rural Health Committee regarding the 25th annual Rural Health Conference, conducted Oct. 7-8, 1987. Almost 500 people attended, representing 41 Tennessee counties.
Committee on Continuing Medical Education	Received a report from the Committee on Continuing Medical Education that the Accreditation Council for Continuing Medical Education Committee on Review and Recognition will likely accredit TMA for another six years.
Impaired Physician Program	Voted to renew the contract of Dr. David Dodd, TMA's medical director for the Impaired Physician Program, for another year.
State Council for Promotion of Health	Voted to support a concept proposed by the State Athletic Trainers Association for development of a state Council on Health and Fitness.
Ad Hoc Committee on Young Physicians Section	Voted to approve funding for a survey of young physicians in Tennessee. Survey data will be reviewed by the Board prior to developing a recommendation on whether or not a young physicians section should be formed within TMA.
CME Program Recognition	Voted to provide a certificate or plaque to those institutions accredited by the TMA for continuing medical education.
Annual Meeting Exhibits	Approved recommended changes to the format of TMA Annual Meeting Exhibits of products and services to include additional food and beverage functions.
Scientific Affairs Committee	Voted to authorize the Scientific Affairs Committee to determine an appropriate reinstatement fee to be charged to those medical specialty societies that fail to complete the required program evaluation for those programs receiving Category 1 CME credit.
Crippled Children's Services	Decided to submit the names of Drs. Lewis Cosby, Johnson City, Timothy W. Thurston, Maryville, and Robert L. Barnes, III, Knoxville, for consideration for appointment to the state Crippled Children's Services Committee.
Medicaid Medical Care Advisory Committee	Decided to submit the names of Drs. Edward W. Reed, Memphis, Luthur A. Beazley, Jr., Nashville, and Marvin G. Gregory, Jr., Nashville, for consideration for appointment to the Medicaid Medical Care Advisory Committee.
Medicaid Formulary Advisory Committee	Decided to submit the names of Drs. Steven Schillig, Nashville, Claude H. Workman, Jr., Memphis, and Cecil E. Russell, Jr., Powell, for consideration for appointment to the Medicaid Formulary Advisory Committee.
1988 Proposed Budget	Accepted the TMA Finance Committee's recommendation and approved a proposed 1988 budget of $1,424,000.

TMA continuing medical education opportunities

The continuing medical education accreditation program of the TMA has full approval by the Accreditation Council for Continuing Medical Education. An accredited institution or organization may designate for Category 1 credit toward the AMA Physician's Recognition Award those CME activities that meet appropriate guidelines. If you wish information as to how your hospital may receive accreditation, write: Director of Continuing Medical Education, Tennessee Medical Association, 112 Louise Ave., Nashville, TN 37203.

IMPORTANT NOTICE

Published in this section are all educational opportunities which come to our attention which might be of interest to our membership. As some of these are very long, full year schedules, and others are detailed descriptions of courses, in order to conserve space, most of them will be published in only one issue of the Journal.

IN TENNESSEE

VANDERBILT UNIVERSITY

Clinical Training Program

Opportunities for advanced clinical education for physicians in various subspecialties have been developed by the School of Medicine and the Division of Continuing Education of Vanderbilt University. The practicing physician, with the guidance of the faculty from the participating department, can plan an individualized program of one to four weeks to meet recognized needs and interests. The experience may include contact with patients; interaction with clinical and full-time faculty, housestaff, and fellows; participation in clinical rounds and diagnostic procedures. There is access to the Medical Center Library with excellent resources. Experience in more than one discipline may be included if time permits.

Eligibility: Licensed physicians in active practice are eligible to apply. *Credit:* AMA Physician's Recognition Award (Category 1) and/or AAFP Continuing Education Accreditation. *Application:* For information and an application contact Marilyn D'Asaro, Division of Continuing Medical Education, Vanderbilt University School of Medicine, CCC-5326 MCN, Nashville, TN 37232, Tel. (615) 322-4030.

Continuing Education Schedule

Jan. 30-Feb. 5	Advances in Medicine—Snowmass Village, Colo.
Feb. 13-19	Advances in Infertility and Reproductive Endocrinology—Snowmass Village, Colo.
Feb. 13-19	Practical Aspects of Diagnostic Radiology/Medical Imaging—Snowmass Village, Colo.
March 5	Early Intervention in Acute MI
March 12-19	The Spine: Current Concepts—Maui, Hawaii
March 17-18	6th Annual Tennessee Conference on Perinatal and Neonatal Care
March 28-29	Tissue-Integrated Prostheses: Aspects of the Prosthodontic Part
April 22-23	Annual Barney Brooks Lecture and the H. William Scott Society
May 6-15	Diving Medicine 1988: Medicine in the Aquatic Environment—Fiji
May 17-22	5th Annual Family Medicine Review
May 27-28	11th Annual Sonography Symposium
June 9-10	2nd Annual Neonatology Conference
June 15-19	Contemporary Medical Imaging V—Destin, Fla.
July 18-22	11th Annual Contemporary Clinical Neurology—Hilton Head Island, S.C.

For information contact Division of Continuing Medical Education, Vanderbilt University School of Medicine, CCC-5326 MCN, Nashville, TN 37232, Tel. (615) 322-4030.

MEHARRY MEDICAL COLLEGE

Extended Continuing Education Program

Arrangements have been made with the following services and departments in the medical school to allow practicing physicians to participate in that service's activities for a period of one day to one week. This program provides an opportunity for physicians to study in depth for a specified period. The schedule of activities is individualized in response to the physician's request by the participating department. The experience includes conferences, ward rounds, audiovisual materials and contact with patients, residents, and faculty.

Fee: \$75 per day or \$275 per five day week of educational experience including lunch and parking at Meharry Medical College. *Credit:* AMA Category 1 of the Physician's Recognition Award, AAFP, American Academy of Family Physicians and Continuing Education Units from Meharry Medical College. *Application:* For information contact Dr. Henry A. Moses, Ph.D., Director of Continuing Education, Meharry Medical College, 1005 D.B. Todd Boulevard, Nashville, TN 37208, Tel. (615) 327-6235.

Continuing Education Schedule

Jan. 12-15	The Scientific and Clinical Basis of Biotherapy in Malignant Diseases—Franklin, Tenn. *Credit:* 28 hours AMA Category 1. *Fee:* \$500.

For information contact Rosalie Avent, Biological Therapy Institute, Hospital Drive, Franklin, TN 37064, Tel. (615) 790-7535.

UNIVERSITY OF TENNESSEE

Continuing Education Schedule

Memphis

Jan. 24-31	Medicine Review '88
Feb. 17-20	Surgical Treatment of Obesity
Feb. 21-26	Update on Genetics, Reproductive Endocrinology and High Risk Pregnancy—Grand Cayman Isl., B.W.I.
Feb. 27-28	Radiology
March 3-6	Pediatric Critical Care
March 10-11	Primary Prevention: Psychopathology

1987 MEMBERSHIP ROSTER TENNESSEE MEDICAL ASSOCIATION

An alphabetical listing of members of the Tennessee Medical Association by county medical society is published as a service to the membership. An asterisk (*) denotes physicians exempt from dues. A dash (—) denotes a student member.

BEDFORD COUNTY MEDICAL SOCIETY

BARNES, DONALD D, SHELBYVILLE
BEAVERS, LANA SHARON, SHELBYVILLE
* CHAMBERS, WALLACE LEE, SHELBYVILLE
* COOPER, ALBERT LEE, SHELBYVILLE
DERRYBERRY, JOHN S, SHELBYVILLE
* FARRAR, TAYLOR, SHELBYVILLE
FELDHAUS, JOSEPH H, SHELBYVILLE
JAYAKODY, FRANK LORENZ, SHELBYVILLE
JOHNSON, SUE PAINE WELCH, SHELBYVILLE
MAGNUSON, CAROL LENT, SHELBYVILLE
MELSON, DANNY LEE, SHELBYVILLE
* MOULDER, GRACE E, SHELBYVILLE
OWNBY, FRED DILLARD, BELL BUCKLE
RICH, EARL FREEMAN, SHELBYVILLE
RICHARDS, AUBREY THOS, SHELBYVILLE
* ROGERS, BENJ CARL, SHELBYVILLE
SELLS JR, SAMUEL P, SHELBYVILLE
SHUTE, ANNE MARIE, SHELBYVILLE
STIMPSON, CHARLES L, SHELBYVILLE
STUBBLEFIELD, CARL THOS, SHELBYVILLE
WOMACK, SARA, SHELBYVILLE

BENTON-HUMPHREYS COUNTY MEDICAL SOCIETY

ALI, MAYSOON SHOCAIR, WAVERLY
ALI, SUBHI DAWUD SUBOH, WAVERLY
BLACKBURN, WM H, CAMDEN
BLANTON, HAROLD L, COURTLAND, AL
BOURNE JR, ROBERT I, CAMDEN
BUTTERWORTH, JOE S, CAMDEN
HAMM, EMILE V, JASPER
HARTLEY, MARK F, WAVERLY
MCCLURE, WALLACE JOE, WAVERLY
SKELTON, M ANGELA, WAVERLY
STONE, RICHARD A, WAVERLY
WALKER, ARTHUR WINFREY, WAVERLY

BLOUNT COUNTY MEDICAL SOCIETY

AGEE, OLIVER KING, MARYVILLE
BEARD, MARVIN ROBISON, MARYVILLE
BELL, W KEN, MARYVILLE
BLANKS, BILLY HARRELL, MARYVILLE
BOLLINGER JR, JOHN A, MARYVILLE
BOWEN, JOHN H, MARYVILLE
BURKHART, PATRICK H, MARYVILLE
CALLAWAY JR, HENRY A, MARYVILLE
CALLAWAY, JAMES MILLER, MARYVILLE
CHRISTOFFERSON, JAMES W, MARYVILLE
CLINE, KIM, LENOIR CITY
CLINE, RICHARD, MARYVILLE
COLEMAN, CHERYL E, MARYVILLE
COWAN, JOHN DAVID, MARYVILLE
CROWDER, CLAY G, MARYVILLE
DORR, DAVID C, KNOXVILLE
ELLINGTON, ERIC PATRICK, MARYVILLE
ELLIOTT, WM EARL, MARYVILLE
ELMORE, DALE B, MARYVILLE
EVANS, SAMUEL D, MARYVILLE
FARRA, FRED T, MARYVILLE
FINNEY JR, RAYMOND A, MARYVILLE
FLICKINGER, TED LAWRENCE, MARYVILLE
GALLAGHER, MICHAEL P, MARYVILLE
GREEN, BRUCE QUINTON, LOUISVILLE
HARALSON III, ROBT HATTON, MARYVILLE
* HARALSON JR, ROBT H, MARYVILLE
HATFIELD, CHAS NEWMAN, MARYVILLE
HAUN JR, LOUIS EUGENE, MARYVILLE
HEINY, JEROME JAMES, MARYVILLE
HENDERSON JR, JOS S, ALCOA
* HENRY, JAMES SPENCER, ALCOA
HOFFMANN, PAUL WILFRIED, MARYVILLE
HOLDER, JAMES THOS, MARYVILLE
HOLMES, GREGORY M, MARYVILLE
HOWARD, CECIL B, MARYVILLE
HUFFMAN, JOHN RAYMOND, MARYVILLE
INGRAM III, JOHN JACKSON, MARYVILLE
ISBELL JR, HOMER L, MARYVILLE
JACKSON, RICHARD EVAN, MARYVILLE
JARVIS, S CRAIG, MARYVILLE
JENKINS, BASIA IRENE M, KNOXVILLE
KAMPERMAN, COLIN LEE, ALCOA
KIEFER, STEPHEN K, MARYVILLE
KINTNER, ELGIN P, MARYVILLE
* LAMBETH, SAML S, MARYVILLE
LAUGHMILLER, ROY W, MARYVILLE
* LENTZ, JULIAN C, FAYETTEVILLE, NC
LEYEN, ROBT F, MARYVILLE
MANDRELL, JOE THOS, ALCOA
MARMON, KENNETH WALDO, MARYVILLE
MCAMIS, JOHN CARL, MARYVILLE
MCCROSKEY, DAVID L, MARYVILLE
MCKINNON JR, NORMAN A, MARYVILLE
MILLARD, JAMES HENRY, MARYVILLE
MINNICH, THOMAS E, FRIENDSVILLE
MOHON, RICKY T, MARYVILLE
MOORE, WILLIAM R, MARYVILLE
MYNATT, RICHARD J, TOWNSEND
NELSON, HENRY SPERRY, MARYVILLE
PERSHING, STEPHEN D, MARYVILLE
PETERSON, MARVIN DEAN, MARYVILLE
* PHELAN, JACK STANISLAUS, MARYVILLE
PITTENGER, JOHN, MARYVILLE
PROFFITT, JAMES NICHOLAS, MARYVILLE
PROFFITT, ROBT DAVID, MARYVILLE
* RAMSEY, BAINARD PERCY, MARYVILLE
RAPER, CHAS ALLEN, MARYVILLE
RICCIARDI, JAMES EDWARD, MARYVILLE
ROBERTS JR, JACK T, MARYVILLE
ROMANS, ALLAN, MARYVILLE
SEATON, ROBERT W, MARYVILLE
SIMPSON JR, OSCAR L, MARYVILLE
SMALLEY JR, J BRYAN, MARYVILLE
SMUCKLER, ALAN LEE, MARYVILLE
SOMMERVILLE JR, LEWIS C, MARYVILLE
THOMPSON, BRYAN BROOKS, MARYVILLE
THURSTON, TIMOTHY WM, MARYVILLE
TOLHURST, GEORGE F, MARYVILLE
TREKELL, MELISSA E, MARYVILLE
VALENTINE, ROBERT S, MARYVILLE
VANDERGRIFF, HARRIS T, MARYVILLE
WEATHERBEE, TAYLOR CARSON, MARYVILLE
WEBB, JOHN V, MARYVILLE
WHITE, RICHARD H, MARYVILLE
YARBOROUGH, JOHN A, MARYVILLE

BRADLEY COUNTY MEDICAL SOCIETY

ALDRICH, WM T, CLEVELAND
ANDERSON, DAVID, CLEVELAND
APPLING, JOHN MORGAN, CLEVELAND
ARNOLD JR, CHAS WM, CLEVELAND
* BATCHELOR, MARVIN R, CLEVELAND
BEASLEY, ROBERT ALAN, CLEVELAND
BENTON III, OLIVER, CLEVELAND
BESING, JOHN WM, CLEVELAND
BOWERS, WILLIAM D, CLEVELAND
BREWER, RANDALL J, CLEVELAND
BRYAN, JOHN MILTON, CLEVELAND
BYERS, GLEN MARSH, CLEVELAND
BYRD, JACK, CLEVELAND
BZIK, PETER, CLEVELAND
CHAFFIN, DAVID C, CLEVELAND
CHAMBERS, JOHN WALLACE, CLEVELAND
CHASTAIN JR, CHALMER, CLEVELAND
CHASTAIN, ALLAN CHALMER, CLEVELAND
CHASTAIN, KENT, CLEVELAND
CLARK, MARC LEWIS, CLEVELAND
COFER, ROBT HARRISON, CLEVELAND
COLEMAN, RONALD S, CLEVELAND
COLLINS, LARRY C, CLEVELAND
DAUBNER, ELIZABETH ALLEN, CLEVELAND
DAUBNER, MICHAEL ALAN, CLEVELAND
DEVANE, JERRY, CLEVELAND
DEVANE, JO LEE, CLEVELAND
DULL, ROBERT, BENTON
DUNCAN, EDDIE NORRIS, CLEVELAND
FORD, DENNIS CLIFFORD, CLEVELAND
GIBSON, DONALD BAKER, CLEVELAND
GOLDMAN, MAURICE SAML, CLEVELAND
GRAFTON JR, EDWIN G, CLEVELAND
HAMBY, DONALD LYNN, CLEVELAND
HAMILTON, HOWARD KEN, CLEVELAND
HARTING, DON C, CLEVELAND
HUGHES, CHAS RICHARD, CLEVELAND
JOHNSON JR, WM FRANK, CLEVELAND
JOHNSON, DANIEL V, CLEVELAND
JOHNSON, WM W, CLEVELAND
JONES JR, FRANK KELLEY, CLEVELAND
KIM, STEVEN, CLEVELAND
KIMBALL, CECIL HARRY, CLEVELAND
KNABB, JAMES, CLEVELAND
KNIGHT, C DEWAYNE, CLEVELAND
KYLE JR, CLYDE A, CLEVELAND
LEE, WM REECE, COPPERHILL
LEISY, MARILYN, CLEVELAND
* LOWE, JAMES CECIL, CLEVELAND
MCKINNEY JR, EDWARD D, CLEVELAND
MCNULTY, JOHN STEPHEN, CLEVELAND
MITCHELL, HAYS, CLEVELAND
MONNIG, JACK ANTHONY, CLEVELAND
MURPHY, BILL H, CLEVELAND
MURPHY, JOHN ALLEN, CLEVELAND
MUTHS, FREDERICK A, CLEVELAND
NEWTON, NICHOLAS, CLEVELAND
OBERG, RICHARD, CLEVELAND
OZAWA, T TED, CLEVELAND
PIERCE, E HARRIS, CLEVELAND
POWELL, JOHN MANLEY, CLEVELAND
PROFFITT, WM I, CLEVELAND
ROBINSON, DONALD EDWIN, CLEVELAND
ROGNESS, JOHN A, CLEVELAND
ROMAINE, CHAS BOYD, CLEVELAND
SCRUGGS, FENTON LEE, CLEVELAND
SHOEMAKER, KENNETH E, CLEVELAND
* SMITH, WM R, HILTON HEAD IS, SC
SNODDY, JANET ELIZABETH, CLEVELAND
STANDRIDGE, JOHN B, CLEVELAND
STONE, JAMES PATTERSON, CLEVELAND
SWART JR, EDWIN GIFFORD, CLEVELAND
TAYLOR, OWEN C, CLEVELAND
THURMAN, JAMES ROBT, CLEVELAND
TILSON, FORREST BLAIN, CLEVELAND
VANCE, DANIEL B, CLEVELAND
YOUNGER, CLYDE P, CLEVELAND

BUFFALO RIVER VALLEY MEDICAL SOCIETY

ALDERSON, CHAS MALCOLM, PARSONS
ANAND, VEENA, HOHENWALD
ANAND, VIRENDER, HOHENWALD
AVERETT, STEPHEN L, LINDEN
BARDEN, LEROY F, LINDEN
* BLENDER, WILLIAM, LINDEN
* COLEMAN, ROBT M, MURFREESBORO
* COOK, WILLIAM N, PRIMM SPRINGS
ELROD, PARKER DAVID, CENTERVILLE
FOSNES, JEFFREY CARL, CENTERVILLE
HOLLADAY, BERTIE L, CENTERVILLE
TURNER JR, GORDON H, LINDEN

CAMPBELL COUNTY MEDICAL SOCIETY

BURRELL, JOHN S, LAKE CITY
CLINE JR, ELIJAH GRADY, LAFOLLETTE
COHEN, THOS LEONARD, LAFOLLETTE
CRUTCHFIELD, JAMES DONALD, LAFOLLETTE
DAY, GEORGE LOUIS, HARROGATE
FARRIS, JAMES CLARENCE, LAFOLLETTE
GILES, JAMES W, LAFOLLETTE
HALL III, RONALD BAKER, LAFOLLETTE
HARTMAN, RONALD D, JELLICO
HWANG, LI-MIN, LAFOLLETTE
ISHAM, CHARLES AUBREY, LAFOLLETTE
JONES, JOHN R, CORBIN, KY
LILLIBRIDGE, SCOTT R, JELLICO
* PRATER, CHAS ALVIN, JELLICO
* SEARGEANT JR, LEE JESS, LAFOLLETTE
SHIH, YIU-FEI, LAFOLLETTE
STAFFORD, WILLIAM LEWIS, JELLICO
THOMPSON, GEO STANLEY, HARROGATE
WALKER, JESSE LEE, JELLICO
WILKENS, CHAS HENRY, JELLICO
WOOD, BURGIN HENRY, LAFOLLETTE

CARTER COUNTY MEDICAL SOCIETY

ARCHIE, DAVID S, JOHNSON CITY
BASSAL, ALY A, ELIZABETHTON
BRONSON, S MARTIN, ELIZABETHTON
BURIK, NICHOLAS P, ELIZABETHTON
CAUDLE, SCOTT OKRINA, ELIZABETHTON
CHAMBERS, GARY R, ELIZABETHTON
CRAIG, JAMES P, ELIZABETHTON
CRUZ JR, TEODORICO P, ELIZABETHTON
DAVIS, FLOYD, BLOWING ROCK, NC
GALLAHER, RICHARD GRANT, ELIZABETHTON
GALLOWAY, RICHARD EUGENE, ELIZABETHTON
GASTINEAU, JERRY LEE, ELIZABETHTON
GRINDSTAFF, ROBERT E, JOHNSON CITY
HERRIN, C BOMAR, JOHNSON CITY
HOPLAND, ARNOLD D, ELIZABETHTON
LAING, BRENT D, ELIZABETHTON
MARTIN JR, RICARDO S, ELIZABETHTON
MAY, FLOYD E, ELIZABETHTON
MAY, W JOYCE, ELIZABETHTON
* PEARSON, ELMER TYLER, ELIZABETHTON
PERRY, EDGAR EUGENE, ELIZABETHTON
SLAGLE, DAVID J, ELIZABETHTON
TAYLOR, TEOFORO STEVE, ELIZABETHTON
WALTER, ROBERT E, ELIZABETHTON
WELLS, CHARLES J, ELIZABETHTON

CHATTANOOGA-HAMILTON COUNTY MEDICAL SOCIETY

ABELL, JAMES E, CHATTANOOGA
ABRAMSON, JEROME H, CHATTANOOGA
* ADAMS JR, JOHN W, CHATTANOOGA
ADCOCK, CHARLES R, SOUTH PITTSBURG
AIKEN, MICHAEL M, CHATTANOOGA
AIKEN, WM PRIGMORE, CHATTANOOGA
AKIN, EDGAR DANL, CHATTANOOGA
ALBRITTON, JOHN THOS, CHATTANOOGA
ALLEN, BILLY JASON, OOLTEWAH
ALLEN, GEORGE E, CHATTANOOGA
ALLEN, L DIANNE, OOLTEWAH
ALPER, CHAS H, CHATTANOOGA
ALVAREZ, RICHARD G, CHATTANOOGA
* ANDERSON, HARRY S, CHATTANOOGA
ANDERSON, STEVEN R, CHATTANOOGA
APYAN, PAUL M, HIXSON

ARNOLD, COLEMAN LEE, CHATTANOOGA
ARNOLD, IRA L, CHATTANOOGA
ATKINSON, JOS SPROTT, CHATTANOOGA
AVERY, JOEL EUGENE, CHATTANOOGA
BACON, STUART PETER, DAYTON
* BALLARD JR, FRED B, SIGNAL MOUNTAIN
BANKS JR, WOODRUFF A, CHATTANOOGA
BANKS, SAML LOUIS, CHATTANOOGA
BAREDDY, SWARNA K, CHATTANOOGA
BAREDDY, VENKATA R R, CHATTANOOGA
BARNES, DAVID R, CHATTANOOGA
BARNETT III, ROBERT M, CHATTANOOGA
BARNETT, FRANCES H, WHITWELL
BARTLETT, DAVID O, CHATTANOOGA
BAUTISTA, JUANCHO C, HIXSON
BEAHM, THOMAS M, CHATTANOOGA
BECHARD, DOUGLAS L, CHATTANOOGA
BERGLUND, ROBERT K, CHATTANOOGA
BINDER, SAML S, CHATTANOOGA
BISHOP, FRANK E, CHATTANOOGA
* BISHOP, WM RUSSELL, LOOKOUT MTN
BLAKE, CHAS ALAN, HARRISON
BLOUNT JR, HENRY C, CHATTANOOGA
BOATWRIGHT, CATHERINE A, CHATTANOOGA
BOATWRIGHT, ROBT W, HIXSON
BOAZ JR, LONNIE ROY, CHATTANOOGA
BOAZ, VALERIE A, CHATTANOOGA
BOEHM, PETER ERIC, CHATTANOOGA
* BOEHM, WALTER EDWARD, CHATTANOOGA
BOEHM, WALTER MICHAEL, CHATTANOOGA
BOISER, ANITA MADRIA, HIXSON
BOISER, ARISTIDES L, HIXSON
BOLINGER, JOHN MCCALLIE, CHATTANOOGA
BONDER, MICHAEL IAN, CHATTANOOGA
BOOKOUT, MARK WILLIAM, EAST RIDGE
BOWDEN JR, HARVEY O, CHATTANOOGA
BOWERS JR, JEMISON O, CHATTANOOGA
BOWERS, ROBT EUGENE, CHATTANOOGA
BOWNDS, CHARLES P, PIKEVILLE
BOXELL, JOHN FREDERICK, CHATTANOOGA
BRACKETT, WM DAVID, CHATTANOOGA
* BRANNEN, FRANK S, CHATTANOOGA
BREMER JR, JOEL LEWIS, CHATTANOOGA
BRICE, CHARLES TERRY, CHATTANOOGA
BRIGHT III, THOMAS C, CHATTANOOGA
BRIMI, JOHN BENJ, HIXSON
BROADSTONE, PAUL A, CHATTANOOGA
BROOKSBANK, RONALD C, CHATTANOOGA
BROWN, HOWARD A, CHATTANOOGA
BROWN, HUGH P, CHATTANOOGA
BROWN, NEIL CHAS, CHATTANOOGA
BRYANT, JOHN FRANK, CHATTANOOGA
BUCHANAN JR, THOS F, CHATTANOOGA
BUCHNER III, EDWARD F, CHATTANOOGA
BUCHNER, WM FRANCIS, CHATTANOOGA
* BULLARD, ARCH H, CHATTANOOGA
* BURKE, JOHN ARTHUR, HILTON HEAD ISL, SC
BURNS, RANDEL PHILLIP, CHATTANOOGA
BUTTRAM JR, WM R, CHATTANOOGA
BUTTRAM, THOS LATHAM, CHATTANOOGA
BUTTRAM, W REES, CHATTANOOGA
CAINE JR, WINSTON P, CHATTANOOGA
CALDWELL, GARY BLAINE, CHATTANOOGA
CALHOUN JR, CALVIN LEE, CHATTANOOGA
CAMPBELL, DONALD ROSS, CHATTANOOGA
CAMPBELL, WILLIAM O'NEAL, CHATTANOOGA
CANNON, DON ALLEN, CHATTANOOGA
* CANNON, GEO MARSHALL, SIGNAL MOUNTAIN
* CANON, MAURICE A, CHATTANOOGA
CARR, MICHAEL GRADY, CHATTANOOGA
CARROLL, RAMON LEONARD, CHATTANOOGA
CAUGHRAN, BENNETT W, CHATTANOOGA
CAUGHRAN, DONALD G, CHATTANOOGA
CH'IEN, LAWRENCE TIEN-TSO, CHATTANOOGA
CHADWICK, DAVID AIREY, CHATTANOOGA
CHAMBERLAIN II, MORROW, CHATTANOOGA
CHANDLER, LUTHER F, HIXSON
CHANDRA, CHANNAPPA, HIXSON
* CHASTAIN, CLEO, SAN ANTONIO, TX
CHENG, TIEN H, CHATTANOOGA
CLARK, MURRELL O, CHATTANOOGA
CLEAVELAND, CLIFTON RANCE, CHATTANOOGA
CLEMENTS, JOEL BENJ, CHATTANOOGA
CODDINGTON, ROBT CHAS, CHATTANOOGA
COFER, JOSEPH B, CHATTANOOGA
COLLINS, DAVID NEWTON, CHATTANOOGA
COLLINS, JOHN RICHARD, CHATTANOOGA
CONN, ERIC HAULEY, CHATTANOOGA
COOK, THOMAS ANDREW, CHATTANOOGA
COOPER, FLOYD C, CHATTANOOGA
COPELAND, CHRISTOPHER S, CHATTANOOGA
COREY JR, JAMES HICKS, CHATTANOOGA
COX, GEO EDWIN, CHATTANOOGA
COX, JOHN MICHAEL, CHATTANOOGA
COX, SUE CLARKE, CHATTANOOGA
CRAFT, PHIL DOUGLAS, CHATTANOOGA
CRAIG, ROBERT E LEE, CHATTANOOGA
CRANWELL, JOHN O, CHATTANOOGA
CRAWLEY JR, JAMES F, CHATTANOOGA
CRAWLEY JR, WILLIAM D, ROSSVILLE, GA
CREECH, ROBERT H, CHATTANOOGA
CREEL JR, JAMES HEATON, CHATTANOOGA
CROWELL, JOHN M, CHATTANOOGA
* CURREY, JOE T, CHATTANOOGA
CURREY, THOS WOODRUFF, CHATTANOOGA
CURTIS, THOS H, CHATTANOOGA
DAGHLIAN, BEDROS D, HIXSON
DAHRLING II, BRUCE E, CHATTANOOGA
DANIELL, MALCOLM BUTLER, CHATTANOOGA
DAVIDSON, DEANNA STARR, CHATTANOOGA
DAVIS JR, JAMES PHILLIP, CHATTANOOGA
DAVIS, CHARLES M, CHATTANOOGA
DAVIS, JAMES WILSON, CHATTANOOGA
DAVIS, JIMMY B, CHATTANOOGA
DAVIS, LARRY W, CHATTANOOGA
DAVIS, TIMOTHY P, CHATTANOOGA
DE RUITER, PETER LOUIS, CHATTANOOGA
DEMOS, ROBT G, CHATTANOOGA
DICKINSON, ELIZABETH B, CHATTANOOGA
DICKSON, RICHARD C, CHATTANOOGA
DODDS, JOS JAMES, CHATTANOOGA
DODSON, DAVID BRYAN, CHATTANOOGA
DOLAN, PATRICK, CHATTANOOGA
* DONALDSON, RICHARD B, CHATTANOOGA
DONALDSON, RICHARD WM, CHATTANOOGA
DOUGLAS, MICHAEL, CHATTANOOGA
DOWELL, WM CURTIS, HIXSON
DOWLEN, STEVEN H, CHATTANOOGA
DRAKE, JAMES ROBT, CHATTANOOGA
DRAKE, ROBERT A, CHATTANOOGA
DRESSLER, STANLEY JAY, CHATTANOOGA
DROCKER, DAVID HABER, CHATTANOOGA
DUCKETT, WILLIAM D, HIXSON
DUFFY, MARY A, CHATTANOOGA
DUGAN, PHILIP JERALD, CHATTANOOGA
DUNCAN, VICTOR A, CHATTANOOGA
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CHRISTENBERRY JR, K W, KNOXVILLE
* CHRISTENBERRY, KENNETH W, KNOXVILLE
* CHRISTIAN, HENRY S, KNOXVILLE
CHRISTIANSEN, DEBORAH J, KNOXVILLE
CLOUD, WM WILEY, KNOXVILLE
COLE, ROBT RELAND, KNOXVILLE
COLLIER JR, ROBT HOYAL, KNOXVILLE
CULLMANN, IRVING REID, KNOXVILLE
COMAS, FRANK VILANOVA, KNOXVILLE
* CONGDON, CHAS C, OAK RIDGE
CONLEY, DEAN RAYMOND, KNOXVILLE
* CONNER, EDWARD D, KNOXVILLE
COOLEY, CAROLINE E, KNOXVILLE
COOPER JR, JOHN HARRISON, KNOXVILLE
COPAS, PLEAS R, KNOXVILLE
COREY, DAVID ANTHONY, KNOXVILLE
COUGHLIN JR, DENNIS, KNOXVILLE
CRAWLEY, ROBERT A, KNOXVILLE
CREUTZINGER, DAVID J, KNOXVILLE
CRUMLEY, JOE C, KNOXVILLE
* CULLUM, JESSE P, KNOXVILLE
CURFMAN, W CORBET, KNOXVILLE
DABBS, RANDAL L, KNOXVILLE
DALTON, MORRIS NORTON, KNOXVILLE
DAVIS, LLOYD CLEVELAND, KNOXVILLE
DE FIORE JR, JOS CHAS, KNOXVILLE
DE LEESE, JOSEPH S, KNOXVILLE
DE PERSIO, RICHARD J, KNOXVILLE
DEMERS, ROBERT G, KNOXVILLE
DENNENY III, JAMES C, KNOXVILLE
* DIDDLE, ALBERT W, KNOXVILLE
DILL, STEPHEN H, KNOXVILLE
DILWORTH, LEE L, KNOXVILLE
DOIRON, CLINT T, KNOXVILLE
* DOMM, SHELDON EDWARD, KNOXVILLE
DORSEY, LARRY, KNOXVILLE
DOUGHERTY JR, JOHN H, KNOXVILLE
DOUGHERTY, ROBT EDWARD, KNOXVILLE
DOWNS, JAMES E, KNOXVILLE
* DRESNER, EVELYN EDITH, KNOXVILLE
DUFFY III, RICHARD N, MARYVILLE
DUFFY, MARY BROCK, KNOXVILLE
* DUKES, JAMES B, KNOXVILLE
DUNCAN JR, RAPHAEL H, KNOXVILLE
* DUNCAN, ORVILLE JACK, KNOXVILLE
EARNEST JR, CHAS R, KNOXVILLE
EARNHARDT, J WILLIAM, KNOXVILLE
EASTHAM, JEROME F, KNOXVILLE
EBENEZER, C S ALBERT, KNOXVILLE
EBERLE, ANDREA J, KNOXVILLE
EISELE, SANDRA A, KNOXVILLE
EISENSTADT, MICHAEL L, KNOXVILLE
ELLENBURG, DONALD T, KNOXVILLE
ELLIOTT, MICHAEL B, KNOXVILLE
ELLIS, ROY C, HARROGATE
* ELY, JAMES B, KNOXVILLE
EMBRY, JERRY J, KNOXVILLE
ENGLAND, W DAVID, KNOXVILLE
ERICKSON, RICHARD JAMES, KNOXVILLE
ERWIN, STANLEY W, KNOXVILLE
ETEZADI-AMOLI, SAEED, KNOXVILLE
EVANS, JOHN HAROLD, KNOXVILLE
FARDON, DAVID FAVREAU, KNOXVILLE
FARRIS, RICHARD KENT, KNOXVILLE
FECHER, MARK P, KNOXVILLE
FELD, NEIL, KNOXVILLE
FERGUSON, JAMES V, KNOXVILLE
FETZER JR, JOHN WOODROW, JEFFERSON CITY
FILCHOCK, JOANNE, KNOXVILLE
FILLMORE, GEO EDWARD, KNOXVILLE
FINELLI, ROBERT EDWARD, KNOXVILLE
* FINER, GEO HARVEY, SARASOTA, FL
FOGLE, RICHARD ALLEN, KNOXVILLE
FOSTER, JERRY M, KNOXVILLE
FOSTER, WILLIAM EDWIN, KNOXVILLE
FRAME, BARRY D, KNOXVILLE
* FREEDMAN, HAROLD D, LENOIR CITY
FREEMAN, COY, KNOXVILLE
FRERE JR, JOHN M, KNOXVILLE
FRY JR, MELLON ALMA, KNOXVILLE
FURR, FRED M, KNOXVILLE
GALIARDI, MARTY P, KNOXVILLE
GALYON JR, FRANK B, KNOXVILLE
GARCIA JR, JOS ISABEL, KNOXVILLE
GARDNER, WM HENRY, KNOXVILLE
GARRETT JR, ALBERT S, OAK RIDGE
GEE JR, GEO LEONARD, KNOXVILLE
GENTRY, ROBT HOMER, KNOXVILLE
GERKIN, DAVID GEORGE, KNOXVILLE
GIBSON, CARL EUGENE, KNOXVILLE
GILBERTSON, ROBT B, KNOXVILLE
GILLESPIE, RICHARD ALLEN, KNOXVILLE
GILREATH, CATHERINE ANN, KNOXVILLE
GIOGIANNI, MARIO G, KNOXVILLE
GITSCHLAG, GARY N, KNOXVILLE
GITSCHLAG, KAMILIA F, KNOXVILLE
GLEAVES JR, JAMES E, KNOXVILLE
GLOVER JR, ABNER M, KNOXVILLE
GLOVER, A MICHAEL, KNOXVILLE
GODWIN, CHAS WAYNE, KNOXVILLE
GOLDMAN, MITCHELL H, KNOXVILLE
* GOODGE, BAYARD D, CONCORD
GOODGE JR, JOSEPH M, KNOXVILLE
GOODELOCK, D STEVENSON, LOUISVILLE
GOUFFON, CHAS ALLEN, KNOXVILLE
GOULD, HOWARD R, KNOXVILLE
GRABEEL, CONRAD LINDSAY, KNOXVILLE
GRAHAM, RANDAL D, KNOXVILLE
GRAY, FRANK BENTON, KNOXVILLE
GREENE, JAMES ALLEN, KNOXVILLE
GREENE, RICHARD W, KNOXVILLE
GREENWOOD, JEFFERY D, KNOXVILLE
GREGSON, GORDON S, KNOXVILLE
GRIFFITH, ROBT CARL, KNOXVILLE
GROSSMAN, ALLAN M, KNOXVILLE
GROVES, ROBERT MCDONALD, KNOXVILLE
GUYTON JR, JAMES R, KNOXVILLE
GYORIK, CATHERINE F, KNOXVILLE
HAASE JR, THEODORE F, KNOXVILLE
HAHN, JAN F, LENOIR CITY
HALL, CEN J, KNOXVILLE
HALL, ROBT EDMUND, KNOXVILLE
HAMPTON, BERT ALLAN, KNOXVILLE
HANNA, WAHID T, KNOXVILLE
HAQ, JAMSHED U, KNOXVILLE
HARAF, FRANK JOS, KNOXVILLE
HARB, JOS W, KNOXVILLE
HARGROVE, R LESLIE, KNOXVILLE
HARRELL, THOMAS G, KNOXVILLE
HARRIS, ROBT WAYNE, KNOXVILLE
HARRISON, JOHN E B, KNOXVILLE
* HARRISON, WM BLAIR, LOUDON
HASSELL, DAVID E, KNOXVILLE
* HAYES JR, TUCKEY J T, KNOXVILLE
HAYS JR, JAMES M, KNOXVILLE
HAYWORTH, RAY MILTON, KNOXVILLE
HECHT, JEFFREY S, KNOXVILLE
* HEINTZELMAN, JOHN H L, SEAL BEACH, CA
HEISER, DON RICHARD, KNOXVILLE
HEMBREE, DOUGLAS KIRBY, KNOXVILLE
HEMPHILL, JAMES LOUIS, KNOXVILLE
HENDERSON, RICHARD WINN, KNOXVILLE
HENRY, BERTRAM ROWE, KNOXVILLE
HETRICK, THOMAS HENRY, KNOXVILLE
HICKS JR, HOWARD KENNETH, KNOXVILLE
* HICKS, HOWARD KENNETH, KNOXVILLE
HIGGINS, THOMAS G, KNOXVILLE
HILL, HUBERT CAWOOD, KNOXVILLE
HITCH JR, JAMES PARKS, KNOXVILLE
HOBART JR, RICHARD LOREN, KNOXVILLE
HOGGE, FREDERICK WM, KNOXVILLE
* HOEY, DAVID FRANCIS, KNOXVILLE
HOGAN, WILLIAM MITCHELL, KNOXVILLE
HOSKINS, JOHN C, KNOXVILLE
* HOSKINS, LEON CUNO, KNOXVILLE
HOVIS, WM MARVIN, KNOXVILLE
* HOWARD JR, G TURNER, KNOXVILLE
HOWE, JOHN W, KNOXVILLE

HUBNER, KARL FRANZ, KNOXVILLE
HUDDLESTON, CHAS IRVING, KNOXVILLE
HUDGENS JR, JAMES F, KNOXVILLE
HUDSON JR, ARNOLD R, KNOXVILLE
* HUGGIN, PERRY M, KNOXVILLE
HURST, FRED ALAN, KNOXVILLE
HUSKEY, LARRY CECIL, KNOXVILLE
HUTCHINS, STEPHEN F, KNOXVILLE
HUTSON, CHAS COMBS, KNOXVILLE
HYATT, HUGH CROCKETT, KNOXVILLE
HYDE, GILBERT L, KNOXVILLE
* IDOL, ENOCH COLVIN, KNOXVILLE
* IRWIN, CLIFTON F, KNOXVILLE
IVENS, MARK YOUNG, KNOXVILLE
JACKSON, ROBERT C, KNOXVILLE
JANZEN, WM ROY, KNOXVILLE
JEFFRIES, GLENN EDWARD, KNOXVILLE
* JENKINS, ASTOR L, KNOXVILLE
JENNINGS, JEFFORY G, KNOXVILLE
JOBSON, KENNETH D, KNOXVILLE
JOHNSON, CLIFFORD, KNOXVILLE
JOHNSON, JANET K, CORDOVA
JOHNSON, JERRY RICHARD, KNOXVILLE
JOHNSON, JOE BREESE, KNOXVILLE
JONES, FRANCIS S, KNOXVILLE
JOST, RICHARD RAYMOND, SPRING CITY
JOURDAN, PAUL LEON, KNOXVILLE
* JOYCE, MARGARET ELIZABETH, KNOXVILLE
JULIUS, CLARK ELDON, KNOXVILLE
KASERMAN, FRED B, KNOXVILLE
KATTINE, ANTHONY ALBERT, KNOXVILLE
KELLY, ARTHUR PAT, KNOXVILLE
* KELSO, HAROLD MILLS, KNOXVILLE
KENNEDY, A GLENN, KNOXVILLE
* KENNEDY, JOHN OLNEY, KNOXVILLE
KESTERSON, GREGG H O, KNOXVILLE
KESTERSON, JOHN E, KNOXVILLE
KHAIROLLAHI, VALI, KNOXVILLE
KHAN, ABDUL H, KNOXVILLE
KILLEFFER, FRED AYRES, KNOXVILLE
KIM, YOO KEUN, KNOXVILLE
KINCAID, GEOFFREY C, KNOXVILLE
KING, IRVIN RAY, KNOXVILLE
KING, JACK DONALD, KNOXVILLE
KIRK JR, CLIFFORD C, KNOXVILLE
* KLEIN JR, VICTOR HILL, KNOXVILLE
KLEIN, CARL JOHN, KNOXVILLE
KLEIN, FREDERICK A, KNOXVILLE
KLIEFOTH III, A BERNHARD, KNOXVILLE
KNOWLING, ROBT EDWARD, KNOXVILLE
KOEFOOT JR, R BRUCE, KNOXVILLE
KRAUSS, STEPHEN, KNOXVILLE
KRISLE III, GEORGE MENEES, KNOXVILLE
KUBOTA, THOMAS T, KNOXVILLE
LACEY III, JOHN W, KNOXVILLE
LAING, WM GAVIN, KNOXVILLE
LASH, ROBT F, KNOXVILLE
LATHAM, KENT EMERSON, KNOXVILLE
LAW JR, WILLIAM M, KNOXVILLE
LAW SR, WILLIAM M, KNOXVILLE
LAZARUS, STEPHEN M, KNOXVILLE
LE BEL, SERGE, KNOXVILLE
LEAHY, MICHAEL DOUGLAS, KNOXVILLE
LEONARD, JOE H, KNOXVILLE
* LESHER, JOHN HAROLD, KNOXVILLE
LESTER, THOMAS EDWARD, KNOXVILLE
LETARO, FRANCIS X, KNOXVILLE
LETHCO, GARY W, KNOXVILLE
LEWIS, GLORIA L, KNOXVILLE
LEWIS, ROBT A, KNOXVILLE
LIGHTER, DONALD E, KNOXVILLE
LIN, KUANG-TZU DAVIS, KNOXVILLE
LINE, FELIX GLEN, KNOXVILLE
LITTLEFIELD, THOMAS R, KNOXVILLE
LONDON, FRANK, KNOXVILLE
LONG, HENRY HEATH, KNOXVILLE
LORCH, VICHIEN, KNOXVILLE
LOWRY, RANDOLPH M, KNOXVILLE
LOWRY, THOS HENRY, KNOXVILLE
LOZZIO, CARMEN BERTUCCI, KNOXVILLE
LUNA, JOE LOUIS, KNOXVILLE
LUTTRELL, ARVELL STANLEY, KNOXVILLE
MACK JR, JOHN W, KNOXVILLE
MACLEAN, RONALD N, KNOXVILLE
MADDOX JR, JOHN R, KNOXVILLE
MADIGAN, ROBT REGIS, KNOXVILLE
MAGGART, MICHAEL L, KNOXVILLE
MALONE JR, EDWARD M, KNOXVILLE
MANCEBO, GERALD L, KNOXVILLE
MANNING, RICHARD O, KNOXVILLE
MANSY, JOSEPH M, KNOXVILLE
MARCY, JOHN SAML, KNOXVILLE
MARSHALL, JOHN HOUSDEN L, KNOXVILLE
MARTIN, CHRIS JOHN, KNOXVILLE
MARTIN, ROBERT O, KNOXVILLE
MASSINGALE, H LYNN, KNOXVILLE
MASTERS, STEVEN BRADLEY, KNOXVILLE
MATHEWS, CARL LESLIE, KNOXVILLE
MAULL, KIMBALL I, KNOVILLE
MAYES, BARRY V, KNOXVILLE
* MAYNARD, MARGARET AGNES, ATLANTA, GA
MCCALLEN, PERRY BOIES, KNOXVILLE
MCCAMMON, CURTIS P, KNOXVILLE
* MCCAMPBELL, BRUCE R, KNOXVILLE
MCCOLLUM, LIONEL O, KNOXVILLE
MCCOY III, WILLIAM JOHN, KNOXVILLE
MCGHEE, WILLIAM EDWARD, KNOXVILLE
MCGINN, LARRY DEAN, KNOXVILLE
MCGINNIS, CARROLL WILLIAM, KNOXVILLE
MCKENZIE JR, ELMER W, KNOXVILLE
MCKENZIE, DONALD KEITH, KNOXVILLE
MCKENZIE, JEROME E, KNOXVILLE
* MCKINNEY, MARION BERRY, KNOXVILLE
MCKISSICK, WILLIAM R, KNOXVILLE
MCMURRY, JOSEPH SEARLE, KNOXVILLE
MCNEELEY, EDWARD TRENT, NORRIS
MCNEELEY, HOWARD B, NORRIS
MCPEAKE III, WILLIAM I, KNOXVILLE
* MCPEAKE, WILLIAM T, LOUDON
MEADOWS, ROBT WALTER, KNOXVILLE
MEISENHEIMER, STEPHEN L, KNOXVILLE
MEYERS, ANTHONY L, KNOXVILLE
MILLER JR, CARTER F, KNOXVILLE
MILLER, CHRISTOPHER A, KNOXVILLE
MILLER, MICHAEL M, KNOXVILLE
MILLER, THOMAS R, KNOXVILLE
MILLER, WM OBED, KNOXVILLE
MIRE, A DEAN, KNOXVILLE
MISRA, SARADA N, KNOXVILLE
MITCHELL SR, ROY B, KNOXVILLE
MITCHELL, DONALD EUGENE, LENOIR CITY
MIXON, WILLIAM R, KNOXVILLE
MOBLEY, JACK MURPHY, KNOXVILLE
MOFFETT, STEVEN R, KNOXVILLE
MONTGOMERY JR, JOHN LEE, KNOXVILLE
MONTGOMERY, JOS TUCKER, KNOXVILLE
MONTGOMERY, ROBERT N, KNOXVILLE
MOON, JOS BENJAMINE, KNOXVILLE
MOORE JR, JOHN DAVID, KNOXVILLE
MOORE JR, MERRILL DENNIS, KNOXVILLE
* MOORE, JOHN DAVID, KNOXVILLE
MOORE, ROBERT SAYLOR, KNOXVILLE
MOORESIDE, DOUGLAS EDWARD, KNOXVILLE
MOREHEAD, LANCE, KNOXVILLE
MORENO, FRANCISCO G, KNOXVILLE
MORGAN, TOMMY E, KNOXVILLE
MORGAN, TRAVIS EUGENE, KNOXVILLE
MORRIS, STEVEN ALLEN, KNOXVILLE
MOSELEY, JAMES F, KNOXVILLE
MOUNGER, EMERSON JAY, KNOXVILLE
MUELLER, ROBT LOUIS, KNOXVILLE
MUMFORD, MARK S, KNOXVILLE
MURPHY, M DIANNE, KNOXVILLE
MURRAY JR, EDWARD LEE, KNOXVILLE
MUSE JR, WM SCOTT, KNOXVILLE
* MUSE SR, WM S, KNOXVILLE
MUTTER, MITCHELL L, KNOXVILLE
MYERS, JAMES DAVID, KNOXVILLE
NATELSON, STEPHEN ELLIS, KNOXVILLE
NEBLETT, JOHN W, KNOXVILLE
NELSON JR, HENRY S, MARYVILLE
NELSON JR, JOHN R, KNOXVILLE
* NELSON JR, WM ALEXANDER, KNOXVILLE
NELSON, BILL M, KNOXVILLE
NELSON, MARK L, KNOXVILLE
NEWTON, KRISTY L, KNOXVILLE
* NICELEY, EUGENE PARK, KNOXVILLE
* NICHOLS, HAZEL MARIE, KNOXVILLE
NORWOOD, CHRISTOPHER W, KNOXVILLE
NOXON, ELVIN B, KNOXVILLE
OBENOUR, RICHARD A, KNOXVILLE
OGDEN, HARRY K, KNOXVILLE
OGLE, HOMER CAMPBELL, KNOXVILLE
OTIS, MICHAEL VAUGHN, KNOXVILLE
OVERHOLT, BERGEIN F, KNOXVILLE
* OVERHOLT, BERGEIN M, KNOXVILLE
OVERHOLT, ROBERT MARION, KNOXVILLE
OWEN, J DAVID, KNOXVILLE
OZDIL, TURAN, KNOXVILLE
PACK, RONALD LYNN, KNOXVILLE
PAINE JR, RAYMOND LEE, KNOXVILLE
PALATINUS, JOS, OAK RIDGE
PAPPAS, SAM GEO, KNOXVILLE
PARK, SOUNG-HO, KNOXVILLE
PARSONS, ROY B, KNOXVILLE
PATIL, VIJAYA R, KNOXVILLE
PATTERSON JR, REESE W, KNOXVILLE
* PATTERSON JR, ROBT F, KNOXVILLE
PATTERSON, FRANCES K, KNOXVILLE
PATTERSON, WM L, KNOXVILLE
PAULSEN, WM ALLEN, KNOXVILLE
* PAYNE, FRANCIS HOMER, KNOXVILLE
PEAGLER, CHAS G, KNOXVILLE
PEARCE, ROBERT E, KNOXVILLE
PEDIGO, RANDALL E, KNOXVILLE
PEEBLES, FRED NEAL, KNOXVILLE
* PENN, JARRELL, KNOXVILLE
PERRY, RONALD HOWARD, KNOXVILLE
* PETERS, H DEWEY, KNOXVILLE
PHELPS JR, PRESTON V, KNOXVILLE
PHELPS, RICHARD W, KNOXVILLE
PHILLIPS, MICHAEL O, KNOXVILLE
PIENKOWSKI, MAREK M, KNOXVILLE
* PIERCE, IRA S, KNOXVILLE
PIERCE, STEVEN FAULKNER, KNOXVILLE
PIERCE, TRUETT H, SNEEDVILLE
PITARO, CECIL F, KNOXVILLE
PLACE, JAMES G, KNOXVILLE
POOL, MICHAEL L, KNOXVILLE
PORTER, E RAYMOND, KNOXVILLE
* POWELL, WM FORREST, KNOXVILLE
* POWERS, BRUCE RANKINS, KNOXVILLE
POWERS, LAURA B, KNOXVILLE
POWERS, WILSON WATKINS, KNOXVILLE
PRESSWOOD, JAMES J, KNOXVILLE
PRIDE, H HAMMOND, KNOXVILLE
PRINCE JR, THOS CHAFER, KNOXVILLE
PRINCE, MARK U, KNOXVILLE
PRINZ, STEPHEN C, KNOXVILLE
PRITCHER, G MARK, KNOXVILLE
* PROSE, JAMES CLINTON, KNOXVILLE
PURVIS, JOHN T, KNOXVILLE
QUILTY, CHARLES L, LOUISVILLE
* RANGE, JOHN W, KNOXVILLE
RANKIN, DAVID M, KNOXVILLE
RAULSTON JR, KENNETH L, KNOXVILLE
RAWSON, FREEMAN L, KNOXVILLE
REATH, DAVID B, KNOXVILLE
REED, STEVEN W, KNOXVILLE
REED, WARREN G, KNOXVILLE
REGESTER JR, ROLLAND E, KNOXVILLE
REID, WILLIAM STUART, KNOXVILLE
REYNOLDS, CHARLES W, KNOXVILLE
* RICHARDS, PAUL D, KNOXVILLE
RIGGINS, BILLY NEWELL, KNOXVILLE
RIMER, RONALD LEE, KNOXVILLE
RIST, TOIVO E, KNOXVILLE
ROBINSON, RICHARD WALTER, KNOXVILLE
ROCHESTER, JOHN CRAWFORD, KNOXVILLE
RODGERS JR, JOHN C, KNOXVILLE
* ROGERS, JERRY RAY, NORFOLK, VA
ROGERS, WM KLAR, KNOXVILLE
ROSE III, RICHARD C, KNOXVILLE
ROWE, BUFORD E, KNOXVILLE
ROWE, CECIL DARRELL, KNOXVILLE
RUDOLPH, BURTON M, KNOXVILLE
RUEFF, DAVID ANTHONY, KNOXVILLE
* RULE III, WILLIAM, KNOXVILLE
RULE, JACK ANDREW, KNOXVILLE
RULE, KENNETH ANDREW, KNOXVILLE
* RULE, KENNETH BOYD, KNOXVILLE
RUSSELL JR, CECIL E, POWELL
RUSSELL, ROBT CLAUDE, KNOXVILLE
RUSSELL, STEPHEN A, KNOXVILLE
RUTH, ALEX, KNOXVILLE
RUTHERFORD JR, CHAS E, KNOXVILLE
RUTHERFORD, KYLE OTIS, KNOXVILLE
RYAN, THOMAS J, SAN ANTONIO, TX
RYLANDS, JOHN CRAIG, KNOXVILLE
* SAFFOLD, JOHN HENRY, KNOXVILLE
SAIN, ROBT LYNN, KNOXVILLE
SANDBERG, RONALD KENNETH, KNOXVILLE
SANDERS, JERRY F, KNOXVILLE
SAWYER, CHRISTOPHER E, KNOXVILLE
SCARIANO JR, JACK F, KNOXVILLE
SCHAFFER, BARRETT A, KNOXVILLE
SCHAUMBURG, EDWIN W, KNOXVILLE
SCHNEIDER, WM JAMES, KNOXVILLE
SCOTT, JOHN CHRISTOPHER, KNOXVILLE
SEALS, ROY LEE, KNOXVILLE
SEATON, DOUGLAS Y, KNOXVILLE
SEGARS, JAMES HUGH, KNOXVILLE
SEMMER, JOHN R, KNOXVILLE
SERRELL, PAUL BURT, KNOXVILLE
SEXTON JR, RICHARD CARR, KNOXVILLE
SEXTON, DAVID G, KNOXVILLE
SEXTON, DAVID HERRON, OAK RIDGE
SEYMOUR, DIGBY GORDON, KNOXVILLE
SHEA JR, WALTER C, LENOIR CITY
SIDDIQI, NASEEMUL HAQ, KNOXVILLE
SIDDIQUI, HAFEEZUL H, KNOXVILLE
* SIENKNECHT, E CHARLES, KNOXVILLE
SILVER, H STEVEN, KNOXVILLE
SIMMONS, JOHN D, KNOXVILLE
SIMMONS, REYNALD T, KNOXVILLE
SIMONS, JON EURIC, KNOXVILLE
SKINNER III, ROLAND L, KNOXVILLE
* SMELTZER, CHAS C, KNOXVILLE
SMITH, BRUCE A, KNOXVILLE
* SMITH, EUGENE BAXTER, KNOXVILLE
SMITH, JOE S, KNOXVILLE
SMITH, LOUIS A, KNOXVILLE
SMITH, MICHAEL G, KNOXVILLE
SMITH, ROBT LLOYD, KNOXVILLE
* SMITH, VERNON I, LOUISVILLE
SMITH, WILLIAM B, NEW ORLEANS, LA
SMITH, WM N, NEW TAZEWELL
SNYDER, EDWARD O, LOUDON
SOLOMON, ALAN, KNOXVILLE
SOSS, SHELDON BARRY, KNOXVILLE
SOWELL, JONATHAN W, KNOXVILLE
SPIEGEL, MARVIN HOWARD, KNOXVILLE
STALLWORTH, WM PARK, KNOXVILLE
* STEVENS, THOS F, KNOXVILLE
STILES JR, JAMES H, KNOXVILLE
STITCHER, RICHARD J, LINCOLN, NE
* STOCKMAN, JOHN MILTON, KNOXVILLE
SUGANTHARAJ, CHRISTIANA R, KNOXVILLE
SULLIVAN JR, THOS ALAN, KNOXVILLE
SULLIVAN, WM ROSS, KNOXVILLE
SUNDAHL, C GERALD, KNOXVILLE
* SWANN JR, WM KIRK, KNOXVILLE
SWEET, JO GORDON, KNOXVILLE
TARWATER, JEAN CATE, KNOXVILLE
TATUM, ROBERT K, KNOXVILLE
TAUXE, EDWARD L, KNOXVILLE
TAYLOR, JAMES WALTER, KNOXVILLE
TAYLOR, KENNETH M, KNOXVILLE
TEAGUE, DALE ALEXANDER, KNOXVILLE
TERRY, WILLIAM F, KNOXVILLE
THOMAS, GARY L, KNOXVILLE
THOMPSON III, JOSEPH L, KNOXVILLE
* THOMPSON, ROBERT G, KNOXVILLE
* TIPTON, WM MARSHALL, KNOXVILLE
TOMPKINS, FORREST G, KNOXVILLE
TONEY III, LEE E, KNOXVILLE
TOYOHARA, HIROSHI, KNOXVILLE
TRAYLOR, THOMAS REID, KNOXVILLE
TREAT, ELMER LAWRENCE, KNOXVILLE
TRENT, BILLY CARL, KNOXVILLE
TRENT, LUCIAN WILLIAMS, KNOXVILLE
* TROTTER JR, GEO MACK, KNOXVILLE
TURNER, JAMES ESPY, KNOXVILLE

* TURNEY, M FRANK, KNOXVILLE
TYLER JR, WM ALEXANDER, KNOXVILLE
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COOPER, CHARLIE WALTER, MEMPHIS
COORS, GEO A, MEMPHIS
COPELAND, GEO D, MEMPHIS
- COPELAND, MARK E, MEMPHIS
CORLEY, GLENNA J, TUSCOLA, IL
CORNELIUS, LELAND RAEBURN, SOUTHAVEN, MS
COSBY, WALTER N, COLUMBUS, MS
COUCH SR, CHARLES E, MEMPHIS
- COUNCE, MICHAEL FORBES, MEMPHIS
COURINGTON, DORIS PAYNE, MEMPHIS
COWAN JR, GEORGE S M, MEMPHIS
COX III, SAM J, MEMPHIS
COX, CLAIR EDWARD, MEMPHIS
- COZART, DAVID, MEMPHIS
- CRANE, PATRICIA G, MEMPHIS
CRAVEN, RUFUS EDGAR, MEMPHIS
CRAWFORD, JOHN D, COLLIERVILLE
CRAWFORD, LLOYD V, MEMPHIS
CRENSHAW JR, ANDREW H, MEMPHIS
CRENSHAW, ANDREW HOYT, MEMPHIS
CRESON JR, THOMAS K, MEMPHIS
CREWS, JOHN T, MEMPHIS
CRISLER JR, HERMAN A, MEMPHIS
- CRISLER, CRISTA L, MEMPHIS
CROCKARELL, JOHN REAMS, MEMPHIS
* CROCKETT JR, ROBT N, MEMPHIS
CROSBY, VIRGIL GLENN, MEMPHIS
- CROUCH, SUSAN F, MEMPHIS
CROWN, LOREN ARTHUR, MEMPHIS
CRUPIE, JOS F, MEMPHIS
CRUTCHER, NANCY, MEMHIS
* CRUTHIRDS, TERRY PARK, MARTIN
CUMMINGS, JOHN M, MEMPHIS
CUMMINS, ALVIN JOS, MEMPHIS
CUNNINGHAM, DAVID LANE, MEMPHIS
CUNNINGHAM, MARK LANE, MEMPHIS
CURLE, RAY EUGENE, MEMPHIS
CURREY, THOS ARTHUR, MEMPHIS
CURTIS, KAREN LOUELLA, MEMPHIS
DANG, LUU HUY, GERMANTOWN
DANIELS JR, WILLIAM WARD, EADS
DAUGHERTY, CHAUNCEY O, MEMPHIS
DAUGHERTY, DAVID R, MEMPHIS
DAVIDSON III, ORIN L, MEMPHIS
- DAVIDSON, WILLIAM R, MEMPHIS
DAVIS JR, JESSE THEO, OXFORD, MS
DAVIS, EDNA M FITZJARREL, MEMPHIS
- DAVIS, ROBERT R, MEMPHIS
DAWOUD, SAMIR RIAD, MEMPHIS
DE MERE, MC CARTHY, MEMPHIS
* DE SAUSSURE JR, R L, MEMPHIS
DE SHAZO, MICHAEL HENRY, MEMPHIS
- DEANA, DANIEL GREGORIO, MEMPHIS
DEATON, WM JERRY, MEMPHIS
DEFRANCO, JOSEPH A, MEMPHIS
- DELEON, DENNIS M, MEMPHIS
DELK, SAM, MEMPHIS
DELLINGER JR, HUBERT L, MEMPHIS
DEMPSEY, BUCKLEY KINARD, GERMANTOWN
DEMPSEY, THOMAS JACKSON, MEMPHIS
DEWANE, JOSEPH C, MEMPHIS
DEWEESE, MELVIN WAYNE, MEMPHIS
DIGAETANO, DOLORES MARIA, MEMPHIS
* DIGGS, LEMUEL WHITLEY, CORDOVA
DILAWARI, RAZA ALI, MEMPHIS
DIRGHANGI, JAYANTA, MEMPHIS
DIRMEYER, PHILLIP HAYS, MEMPHIS
DISMUKE, STEWART EDWARDS, MEMPHIS
DISNEY, JERE MICHAEL, MEMPHIS
DOBSON, JOHN M, MEMPHIS
DODD, RICHARD A, MEMPHIS
DODGE, HERBERT SHUBERT, MEMPHIS
DONAHUE, DAVID J, MEMPHIS
- DOOLEY, PHILIP M, MEMPHIS
DORIAN, JOHN BERNARD, MEMPHIS
- DOUGLAS, ANDREA M, MEMPHIS
- DOWLING, CARRIE L, MEMPHIS
* DOWLING, CHAS VICTOR E, MEMPHIS
DRAGUTSKY, MICHAEL STEVEN, MEMPHIS
DRAKE, ARNOLD MANNAS, MEMPHIS
DRENNING, PAUL THOMAS, MEMPHIS
DREWRY JR, RICHARD DANL, MEMPHIS
- DRINNON, WILLIAM D, MEMPHIS
* DU BARD, MORTON LEE, MEMPHIS
DUBERSTEIN, LARRY EDWIN, MEMPHIS
DUCKWORTH, JOHN KELLY, MEMPHIS
DUCKWORTH, NANCY C H, MEMPHIS
DUGDALE, MARION, MEMPHIS
DUGGIRALA, VIJAYA L, GERMANTOWN
DUKE, DON DE WINDLE, MEMPHIS
DUKE, ROBERT AUBREY, MEMPHIS
DUNAVANT JR, WM DAVID, MEMPHIS
* DUNAVANT, WM DAVID, MEMPHIS
DUNAWAY, DAN ALEXANDER, MEMPHIS
DUNCAN, JERALD MARK, MEMPHIS
DUNCAN-CODY, BARBARA A, MEMPHIS
- DUNN, MICHAEL F, MEMPHIS
DURFEY, JOHN QUINCY, MEMPHIS
- DYCUS, DOUG C, BARTLETT
EASON, HAMEL BOWEN, MEMPHIS
EASON, LESLIE EDMUND, MEMPHIS
ECONOMIDES, NICHOLAS-JOHN, MEMPHIS
EDMONSON, ALLEN S, MEMPHIS
EDWARDS, MARK S, MEMPHIS
EDWARDS, NEIL R, MEMPHIS
EDWARDS, OLGA P, MEMPHIS
EGGERS, FRANK M, MEMPHIS
ELFERVIG, JOHN LARS, MEMPHIS
- ELIAS, CAROLINE K, MEMPHIS
- ELIAS, DAVID BRIAN, MEMPHIS
ELLIOTT, RODNEY GORHAM, MEMPHIS
ELLIS JR, JAMES LEE, MEMPHIS
ELTORKY, MAHMOUD ALI, MEMPHIS
EMERSON, DONALD STEWART, MEMPHIS
EMMETT, JOHN ROY, MEMPHIS
- ENDSLEY, JOHN, MEMPHIS
ENGELBERG, JERRY, MEMPHIS
ENNIS, RICHARD LYN, MEMPHIS
ENSOR, JAMES K, GERMANTOWN
- ENTMAN, HOWARD, MEMPHIS
* ERICKSON, CYRUS CONRAD, MEMPHIS

* ETTELDORF, J N, MEMPHIS
* ETTMAN, IRVING KELSEY, MEMPHIS
* EVANS, MILTON LEE, MEMPHIS
* EVERETT JR, BENNETT E, MEMPHIS
FABIAN, TIMOTHY CHARLES, MEMPHIS
FALVEY, WILLIAM DAVIS, MEMPHIS
FANCHER, WILLIAM H, MEMPHIS
FAQUIN, CORNELL CHAS, MEMPHIS
FARA, LISA F A, MEMPHIS
FARLEY, HAROLD G, MEMPHIS
* FARRAR, TURLEY, MEMPHIS
FARROW JR, C CRESTON, MEMPHIS
- FARROW, ARLEEN C, MEMPHIS
FAULKNER, WM LAWRENCE, MEMPHIS
FEILD, JAMES RODNEY, MEMPHIS
* FEINSTEIN, HAROLD, MEMPHIS
FERGUSON, JOHN MITCHELL, MEMPHIS
- FERRELL III, C LEE, MEMPHIS
FERRELL, THADDEUS HAGAN, MEMPHIS
FIDLER JR, WM JONAS, MEMPHIS
FINK, ROBERT DAVID, MEMPHIS
FIORANELLI, RAYMOND JAMES, MEMPHIS
FISHER JR, JOSEPH N, MEMPHIS
* FISHER, DANL F, MEMPHIS
FISHER, ROBT MOORE, MEMPHIS
FITCH, SARAH JEAN, MEMPHIS
FLANAGAN, JAMES BARRY, MEMPHIS
FLANAGAN, WILLIAM H, MEMPHIS
FLANARY, JAMES S, COLLIERVILLE
FLEMING, IRVIN DURANT, MEMPHIS
FLEMING, JAMES CHRISTIAN, MEMPHIS
FLEMING, JULIAN GLENN, MEMPHIS
- FLEMING, RICHARD A, MEMPHIS
FLINN JR, GEO SHEA, MEMPHIS
FLINN, CARL EDWIN, MEMPHIS
FLORENDO, NOEL TADIAR, MEMPHIS
FLOWERS, WM PARKS, MEMPHIS
FONER, MAX, MEMPHIS
FONG, TERRY, GERMANTOWN
- FOROPOULOS, JOHN E, MEMPHIS
FORTUNE, JAMES EVERETT, MEMPHIS
FOUNTAIN JR, FRANCIS F, MEMPHIS
FOWLER, TOMMY S, MEMPHIS
- FOWLKES, THOMAS D, MEMPHIS
- FRAGETTA, JAMES E, MEMPHIS
FRANCIS JR, HUGH, MEMPHIS
FRANCISCO, JERRY THOS, MEMPHIS
FRANKLIN, EDGAR R, MEMPHIS
FRANKUM, CHAS EUGENE, MEMPHIS
FREEMAN, BARNEY LYNN, MEMPHIS
- FREEMAN, JAMES, MEMPHIS
FREEMAN, JERRE MINOR, MEMPHIS
* FRENCH, WM E, OLIVE BRANCH, MS
FREYTES, CESAR D, MEMPHIS
FRIEDMAN, HARRY, MEMPHIS
- FROMKE, MICHAEL D, MEMPHIS
* FUSTE, RICARDO R, KEY BISCAYNE, FL
FUTRELL, THOMAS WALTER, MEMPHIS
* GADBERRY, EUGENE WARNER, MEMPHIS
GAILLARD, THADDEUS B, MEMPHIS
GAINES, KENNETH J, MEMPHIS
GALINDEZ, TELMO, MEMPHIS
GALYEAN, JAMES R, MEMPHIS
GALYON, JAMES THEODORE, MEMPHIS
GAMMILL, STEPHEN LANE, MEMPHIS
GANT, LINDA L, MEMPHIS
GARBARINI JR, JOS C, MEMPHIS
GARDNER JR, LAWRENCE G, MEMPHIS
GARDNER, JOHN HARVEY, MEMPHIS
GARRETT, HARVEY E, MEMPHIS
GARRETT, RICHARD HENRY, MEMPHIS
GAVANT, MORRIS LEONARD, MEMPHIS
* GAY, JAMES R, LAKEWOOD, PA
GAYDEN, JOHN D, MEMPHIS
GEHI, MOHAN M, MEMPHIS
* GEHORSAM, ELSBETH, MEMPHIS
GELFAND, MICHAEL S, MEMPHIS
GEORGE, JAMES W, MEMPHIS
GEORGE, LEWIS WATSON, MEMPHIS
GEORGE, MORRIS, MEMPHIS
GEORGE, WILBURN E, MEMPHIS
GERALD, BARRY ELMO, MEMPHIS
GESHKE, TERRENCE EDWARD, MEMPHIS
GETTELFINGER, THOMAS C, MEMPHIS
GHIDONI, JOHN JOSEPH, MEMPHIS
GILLULY, JOHN JOS, MEMPHIS
* GINN, BOBBY H, MEMPHIS
* GISH, GEO EDWARD, MEMPHIS
GIVENS, JAMES ROBT, MEMPHIS
GLADDING, THOS CONGDON, MEMPHIS
GLAZER, LOUIS, MEMPHIS
GLOTZBACH, RAYMOND E, MEMPHIS
GOOSEY, WM COLE, MEMPHIS
GOKTURK, TURGUT KEMAL, MEMPHIS
GOLD, ROBERT E, MEMPHIS
* GOLDBERG, FRED A, MEMPHIS
GOLDEN, GERALD S, MEMPHIS
GOLDHAMMER, PHILLIP, MEMPHIS
GOLDIN, MELVIN LESTER, MEMPHIS
GOOCH, JERRY BURTON, MEMPHIS
GOODMAN JR, THOMAS F, MEMPHIS
GOODMAN, JACK A, MEMPHIS
GOODMAN, RALPH C, MEMPHIS
GORDON, TIMOTHY EDWARD, MEMPHIS
GORLINE, WILLIAM JAMES, MEMPHIS
GOTTEN JR, NICHOLAS, MEMPHIS
* GOTTEN, HENRY BRAGG, MEMPHIS
* GOTTEN, NICHOLAS, MEMPHIS
GOURLEY, ROBT DUNSEITH, MEMPHIS
GRAGG, G WINSTON, MEMPHIS
* GRAGG, WILFORD H, MEMPHIS

GRATZ JR, JOHN FISHER, MEMPHIS
GRAVES JR, LESTER R, MEMPHIS
GREEN JR, JAMES BUTLER, MEMPHIS
GREEN, PHILLIP EDWARD, MEMPHIS
GREENWELL, THOMAS D, MEMPHIS
- GREGG, SPENCER D, MEMPHIS
- GREIG, THOMAS WEST, MEMPHIS
GRIFFIN, DANIEL EUGENE, MEMPHIS
GRISE, JERRY WADE, MEMPHIS
GROBMYER III, ALBERT JOS, MEMPHIS
* GROBMYER JR, ALBERT JOS, MEMPHIS
GROGAN JR, FRED T, MEMPHIS
GROSS, CHAS WAYNE, MEMPHIS
GROSSMAN, RONALD K, MEMPHIS
- GUIDI, JOHN F, MEMPHIS
- HAGAN, JOHN B, MEMPHIS
* HAIMSOHN, JAMES S, MEMPHIS
HAJGHASSEMAL, MEHRDOKHT, MEMPHIS
HALFORD III, HOLLIS H, MEMPHIS
HALFORD JR, HOLLIS H, MEMPHIS
HALFORD, JACK RICHARD, MEMPHIS
HALL, JOHNNIE CAMERON, MEMPHIS
* HALL, SYLVIA A, MARTIN
* HALL, VONNIE ARTESIA, MEMPHIS
* HALLE, MARGARET J A, MEMPHIS
- HAMAD, OMAR L, MEMPHIS
HAMILTON, EMILY THOMAS, MEMPHIS
HAMILTON, RALPH F, MEMPHIS
HAMILTON, RALPH S, MEMPHIS
HAMILTON, WM THOS, MEMPHIS
HAMLETT III, JAMES M, MEMPHIS
- HAMNER, H WENTZELL, MEMPHIS
* HAMSHER, JOHN B, MEMPHIS
HANDORF, CHARLES RUSSELL, MEMPHIS
HANISSIAN, ARAM S, MEMPHIS
HANNIFIN, JAMES F, COVINGTON
HARDY, F OLIVER, MEMPHIS
* HARELL, MOSHE, MEMPHIS
HARRELL, ETHEL ASHTON, MEMPHIS
HARRINGTON, OSCAR B, MEMPHIS
HARRIS, BUFORD TERRELL, MEMPHIS
HARRIS, JOHN JOEL, MEMPHIS
HARTNESS, WILLIAM OWEN, MEMPHIS
* HARWELL JR, CARL M, MEMPHIS
HASEN JR, HOWARD B, MEMPHIS
HASEN, HOWARD B, MEMPHIS
HATCH JR, FRED E, MEMPHIS
HAWKES, ALFRED KENNETH, MEMPHIS
* HAWKES, C DOUGLAS, MARCO, FL
* HAWKES, JEAN MURRAY, MARCO ISLAND, FL
HAY, CYRIL LEON, MEMPHIS
HAYES, WM TIMOTHY, MEMPHIS
HAYKAL, RADWAN F, MEMPHIS
HAYS, RACHAEL ANN, ARLINGTON
HEAD, THOMAS GLENN, MEMPHIS
HELLMAN, MICHAEL D, MEMPHIS
HENARD, DONALD CLAUDE, MEMPHIS
HENDRIX JR, JAMES H, MEMPHIS
HENRY, LOUIE C, MEMPHIS
HERNDON JR, BRUCE WAYNE, MEMPHIS
HERRERA, FERNANDO A, MEMPHIS
HIATT, ROGER LEW, MEMPHIS
HICKERSON, WILLIAM L, MEMPHIS
HICKEY JR, HOMER DAVID, MEMPHIS
HIGDON, DENNIS ALAN, MEMPHIS
HIGGINBOTHAM, THOS WAYNE, MEMPHIS
* HIGLEY, GEO BRAINARD, MEMPHIS
HILL, FONTAINE S, MEMPHIS
* HILL, JAMES MARK, MEMPHIS
HILL, JOHN ROY, MEMPHIS
HILSENBECK JR, JOHN ROBERT, MEMPHIS
HINES III, ELBERT E, MEMPHIS
HINES, LEONARD HARVEY, MEMPHIS
HIXSON, SHERMAN D, MEMPHIS
HODGES, JOHN MC IVER, MEMPHIS
- HODGKISS, THOMAS D, MEMPHIS
HOFFMAN JR, WALTER K, MEMPHIS
HOLCOMB, RANDALL L, MEMPHIS
HOLLAPAUGH, ROBT STERLING, MEMPHIS
HOLLAND, NANCY ELIZABETH, MEMPHIS
HOLLIDAY, THOS LINTON, MEMPHIS
HOLLINS, JACK LYNN, MEMPHIS
HOLLIS, DAVID O, MEMPHIS
HOLLOWAY JR, DAVID HOYT, MEMPHIS
* HOLMES, JAMES ELMORE, MEMPHIS
HOLMES, JOHN PIERCE, MEMPHIS
HOLMES, PERRY DON, MEMPHIS
HOLT, EDWIN M, MEMPHIS
HOLT, HUEY THOS, MEMPHIS
HOOD, STEPHEN THOS, MEMPHIS
HOPKINS, JACK T, MEMPHIS
- HOPPER, J BRAIN, MEMPHIS
HORNE, ARTHUR E, MEMPHIS
HORTON, GLENN EDWARD, MEMPHIS
* HOTCHKISS, HUBERT LEECH, BRENTWOOD
* HOUSHOLDER, CHAS H, MEMPHIS
* HOUSTON, JOHN L, MEMPHIS
HOWARD, RUSSELL E, MEMPHIS
HOWSER, JOHN PATTON, MEMPHIS
HUANG, SHANG PO, MEMPHIS
HUBBARD, RONALD EUGENE, MEMPHIS
HUBBERT, CHAS HUGHES, LUBBOCK, TX
HUDSON, JOS STALM, MEMPHIS
HUFFMAN, JOHN DAVID, MEMPHIS
* HUGHES JR, FELIX A, MEMPHIS
HUGHES, ALLEN HOLT, MEMPHIS
* HUGHES, JAMES GILLIAM, MEMPHIS
* HUGHES, JOHN DAVIS, MEMPHIS
* HUGHES, MAX, MEMPHIS
HUGHES, ROBT RULE, MEMPHIS
HUGHES, THOMAS ARTHUR, MEMPHIS

HUGHEY, JOHN R, MEMPHIS
HUMMEL, JOHN VERNON, GERMANTOWN
HUMPHREYS, ROBERT A, MEMPHIS
HUNT, JAMES CALVIN, MEMPHIS
HUNTER, SAML E, MEMPHIS
HUTCHINS, CHAS EDWARD, MEMPHIS
HUTCHINS, LINDA FAYE L, MEMPHIS
- HUTCHISON, TIM, MEMPHIS
IACOBELLI, JUAN WEBER, CORDOVA
* IJAMS, JOE HARTLEY, MEMPHIS
ILABACA, PATRICIO A, MEMPHIS
* INGRAM, ALVIN JOHN, MEMPHIS
JABBOUR, C EUGENE, MEMPHIS
JABBOUR, J T, MEMPHIS
JACKSON, ROBERT LEWIS, MEMPHIS
JACKSON, THOS M, MEMPHIS
JACOBS, ARTHUR ELLIOTT, MEMPHIS
JALLEPALLI, PANDURANGA, MEMPHIS
JAMES, HAL PEARSON, MEMPHIS
- JAMES, THOMAS R, TALLAHASSEE, FL
JARRED, LOIS KATHERINE, MEMPHIS
JARRETT JR, CHAS LESLIE, MEMPHIS
JAUCHLER, GERARD W, MEMPHIS
JEAN-PIERRE, ANTOINE, MEMPHIS
JENKINS, GEORGE W, MEMPHIS
JENKINS, JON CALVIN, MEMPHIS
JENNINGS, DAVID KEITH, MEMPHIS
- JENSEN, JOSEPH C, MEMPHIS
JERKINS, GERALD RAY, MEMPHIS
JEROME, ANTHONY PAUL, MEMPHIS
JOE, PENN DOORK, MEMPHIS
JOHNSON, JAMES GIBB, MEMPHIS
JOHNSON, JOHN WILCOX, MEMPHIS
JOHNSON, LARRY HOLLIDAY, MEMPHIS
JOHNSON, RONALD JACKSON, MEMPHIS
JONES JR, SIDNEY D, MEMPHIS
* JONES, ALBERT MITCHELL, MEMPHIS
JONES, JOE PAUL, MEMPHIS
JONES, R LOBY, MEMPHIS
JONES, ROBT RILEY, MEMPHIS
JONES, WESLEY EARL, MEMPHIS
JORDAN, OAKLEY C, MEMPHIS
- JORDAN, SHELLEY J, MEMPHIS
- JUDGE, DAVID ALLAN, MEMPHIS
* JULICH, ARTHUR WILSON, MEMPHIS
JUSTIS, E JEFF, MEMPHIS
KAHN, SHERMAN ELLIOT, MEMPHIS
- KAISER, GREGG M, MEMPHIS
KANG, ANDREW HO, MEMPHIS
KAPLAN, EDWARD STEVEN, MEMPHIS
KAPLAN, JERRY, MEMPHIS
KAPLAN, ROBT JOEL, MEMPHIS
KAPLAN, STANLEY BARUCH, MEMPHIS
KAPUR, HARI S, MEMPHIS
KASSLES WAHID, LAILA, MEMPHIS
* KASSELBERG, LYMAN A, MEMPHIS
- KATO, DANIEL T, MEMPHIS
- KATZ, PAUL JAY, MEMPHIS
KAVANAGH, KEVIN T, MEMPHIS
KELLERMANN, ARTHUR L, MEMPHIS
KELLETT, GARY LEON, MEMPHIS
KELLEY, BOBBY JERALD, MEMPHIS
* KELLY, ERNEST GEO, MEMPHIS
KELLY, RICHARD T, MEMPHIS
- KEMP, CLAN R, MEMPHIS
KENDRICK JR, WILLIAM RILEY, MEMPHIS
- KENNEDY, A FRANKLIN, MEMPHIS
KERLAN, ROBT ASHLEY, MEMPHIS
* KESSLER, HENRY G, MEMPHIS
KHANDEKAR, ALIM, MEMPHIS
KHANDEKAR, SOPHIA HAQUE, MEMPHIS
KHURI, RADWAN R, MEMPHIS
KIEFER, PATSY R, MEMPHIS
KILEDJIAN, VARTKES, MEMPHIS
KIMBALL, NOAH PEADEN, MEMPHIS
- KIMSEY, FRANK C, MEMPHIS
KIMZEY, GARY, MEMPHIS
KING, BILLY W, MILLINGTON
* KING, CHAS MACK, MEMPHIS
* KING, JOHN C, MEMPHIS
KING, PAUL, MEMPHIS
KING, TRUMAN FRANKLIN, MEMPHIS
KING, WILLIAM SCOTT, MEMPHIS
KINGTON, JOHN MICHAEL, MEMPHIS
KINNARD, JENNIFER J, MEMPHIS
KIRKPATRICK, ROBT DEAN, MEMPHIS
KISABETH, ROBERT M, MEMPHIS
KISHER, RICHARD H, MEMPHIS
KITABCHI, ABBAS EQBAL, MEMPHIS
- KLARICH, KENA T, MEMPHIS
KLEIER JR, ERNEST BOBBY, MEMPHIS
KLINE, ROBT PAUL, MEMPHIS
* KLOTZ, WM F, MEMPHIS
KNIGHT, WILLIAM H, MEMPHIS
KNOTT, DAVID HOWARD, MEMPHIS
- KNOWLTON, SARAH, MEMPHIS
- KNOX III, GEORGE P, MEMPHIS
- KNOX JR, ROBERT L, MEMPHIS
KNOX, ROBT L, MEMPHIS
KOJA, ABED A, MEMPHIS
KOLEYNI, ASGHAR, MEMPHIS
* KOONCE, MARSHALL LYNN, MEMPHIS
* KOSSMANN, CHAS E, MEMPHIS
* KRAUS, ALFRED PAUL, MEMPHIS
KRAUS, GORDON JEROME, MEMPHIS
KRAUS, MELVIN M, MEMPHIS
KRAUS, ROBERT M, MEMPHIS
KRETH, TIMOTHY KERWIN, MEMPHIS
KRISLE JR, JOE RICHARD, MEMPHIS
KROETZ, FRANK WM, MEMPHIS
KRONENBERG, JOEL I, MEMPHIS

KULP, ROY, MEMPHIS
- KURTZ, BRYAN R, NASHVILLE
* KUYKENDALL JR, NATHANIEL, MEMPHIS
* KUYKENDALL, CARY M, MEMPHIS
- KY, HENRY, GERMANTOWN
KYLE, JOS WARREN, MEMPHIS
- LACKEY JR, H LEBRUN, MEMPHIS
LAMAR JR, LUCIUS M, MEMPHIS
LAND, MACK A, MEMPHIS
* LANDSEE, CARL GEO, MILLINGTON
LANDY, STEPHEN HALL, MEMPHIS
LANGFORD JR, C THOMAS, MEMPHIS
LANGSDON, PHILLIP ROYAL, MEMPHIS
LANKFORD, WM ALEXANDER, MEMPHIS
LARIMER, PERRY JAMES, MEMPHIS
LARKIN, CHARLES NEWTON, MEMPHIS
- LARUSSA, RACHEL I, MEMPHIS
LASTER JR, ROBT EUGENE, MEMPHIS
LATHRAM JR, MARVIN W, MEMPHIS
LAUGHLIN JR, ALBERT E, MEMPHIS
* LAUGHLIN SR, ALBERT E, MEMPHIS
LAVELLE JR, HERMAN G, MEMPHIS
LAVELLE, DAVID G, MEMPHIS
- LAWHORN, DAVID WOOD, CHATTANOOGA
LAWRENCE, JESSE ALVAH, MEMPHIS
LAWSON, ROBT EDWARD, MEMPHIS
LAWSON, RONALD D, MEMPHIS
LAZAR, EDWARD HARRY, MEMPHIS
LAZAR, RANDE H, MEMPHIS
LEBOVITZ, M A, MEMPHIS
LEE, LING HONG, MEMPHIS
LEE, SIDNEY REAVES, MEMPHIS
* LEFKOVITS, AARON M, DEERFIELD BEACH, FL
LEMMI, HELIO, MEMPHIS
LEUNG, RICHARD K F, MEMPHIS
LEVENTHAL, MARVIN R, MEMPHIS
LEVINSON, MICHAEL JAY, MEMPHIS
LEVITCH, MELVYN ABRAHAM, MEMPHIS
LEVY, JOE S, MEMPHIS
LEWIS, MYRON, MEMPHIS
* LEWIS, PHILIP M, MEMPHIS
LIEBERMAN, GERALD J, MEMPHIS
LIEBERMAN, PHILLIP LOUIS, MEMPHIS
LIGHT, WILLIAM HARRY, MEMPHIS
LINDER, HILARY FRANCIS, MEMPHIS
LINDERMUTH, JOHN R, MEMPHIS
LING, FRANK W, MEMPHIS
* LIPSCOMB, ALYS H, MEMPHIS
LIPSEY, GEO GARTLEY, MEMPHIS
LITCH JR, MELVIN, MEMPHIS
LITTLE JR, WILLIAM R, MEMPHIS
* LIVERMORE JR, GEO R, MEMPHIS
* LOCKWOOD JR, DUDLEY G, HERNANDO, MS
* LONG, CHAS EDWARD, MEMPHIS
LONG, DIANE M, MEMPHIS
LONG, THOMAS E, MEMPHIS
LONG, WILLIAM E, MEMPHIS
LOUGHEED, JOS C, MEMPHIS
LOVE, VARNA MAE PEYTON, MEMPHIS
LOVEJOY, GEO S, MEMPHIS
- LOVELACE JR, JERRY LEE, MEMPHIS
LOVELESS, SCOTT BARCLAY, MEMPHIS
LOVING, MARTHA A, MEMPHIS
- LU, WILLIAM YUAN HAI, MEMPHIS
LUNCEFORD, TRAVIS E, MEMPHIS
LUTHER, ROBT WAYNE, MEMPHIS
- LYLE, MARY M, MEMPHIS
LYNCH, MICHAEL HARDY, MEMPHIS
LYNCH, PENNY BETH, MEMPHIS
MABRY JR, EDWARD HAYS, MEMPHIS
* MABRY, EDWARD HAYS, MEMPHIS
MACHIN, JAMES ELLIOTT, MEMPHIS
MACKEY, WM FREDERICK, MEMPHIS
MADDOX JR, H BENJAMIN, MEMPHIS
MADDUX, HOLT BENJ, MEMPHIS
MADUSKA, ALBERT LOWELL, MEMPHIS
MAGILL, HUBERT LYNN, MEMPHIS
* MAGUDA, THOS ANDREW, VENICE, FL
MAGUIRE, JAMES K, MEMPHIS
MAHESH-KUMAR, A P, MEMPHIS
MAHFOOD, SAADE S, MEMPHIS
MAIJUB, AMADO GABRIEL, MEMPHIS
* MALONE II, WM B, MEMPHIS
MANDELL, ALAN I, MEMPHIS
MANGIANTE, EUGENE C, MEMPHIS
MANN, JAMES ALAN, MEMPHIS
MANUGIAN, ARSEN, MEMPHIS
MARIENCHECK, WM IRVIN, MEMPHIS
MARKER, HOWARD WM, MEMPHIS
* MARKLE, PHILIP METRIC, MEMPHIS
MARSHALL, DANIEL P, MEMPHIS
MARSHALL, GAILEN D, MEMPHIS
MARSHALL, MICHAEL RALPH, MEMPHIS
* MARTEN, GEO W, MEMPHIS
MARTIN JR, HENRY FRANKJ, MEMPHIS
MARTIN, DANIEL C, MEMPHIS
- MARTIN, FREDERICK A, MEMPHIS
MARTIN, ROY WAYNE, MEMPHIS
* MASON, WM W, HIGHLAND, NC
MASSIE, JAMES D, MEMPHIS
- MATHES, CATHY L, KNOXVILLE
MATHES, GORDON LAWRENCE, MEMPHIS
- MATHIS, CHRISTOPHER L, MEMPHIS
* MATTHEWS, OLIVER S, MEMPHIS
* MAYER, RAYMOND FRANKLIN, MEMPHIS
* MAYFIELD, LEROY H, MEMPHIS
MAYS, KIT SANFORD, MEMPHIS
MCAFEE, JAMES EARL, MEMPHIS
MCBURNEY, ROBERT POWERS, MEMPHIS
* MCCALL, JOHN WILLIAM, MEMPHIS
MCCARTER JR, JOHN G, MEMPHIS
MCCAUGHAN JR, JOHN JOE, MEMPHIS
MCCLOY, RANDOLPH M, MEMPHIS
MCCLURE, JAMES G, MEMPHIS
* MCCOOL, D C, MEMPHIS
MCCORMACK, HAROLD ARTHUR, MEMPHIS
- MCCULLOUGH, CAROL H, MEMPHIS
MCDANIEL III, CARTER E, MEMPHIS
* MCDANIEL, E F, MEMPHIS
- MCELROY JR, AUBREY D, MEMPHIS
MCEWAN JR, ROBERT C, MEMPHIS
MCGEE, JESSE EDWARD, MEMPHIS
MCGEE, JOHN LAWRENCE, MEMPHIS
MCGOWAN, LESLIE R, MEMPHIS
MCGREW III, FRANK A, MEMPHIS
MCKENZIE, EUGENE EATON, MEMPHIS
* MCKINNEY, JAMES W, MEMPHIS
- MCKNIGHT, DONALD T, MEMPHIS
MCLARTY, ALEXANDER M, MEMPHIS
MCLARTY, BARNEY ESTES, MEMPHIS
MCLEMORE JR, THOMAS E, MEMPHIS
MCLENDON, RICHARD ELLISON, GERMANTOWN
MCSWAIN, HAROLD M, MEMPHIS
MERCER, CHARLES WAYNE, MEMPHIS
MERIWETHER III, THOS W, MEMPHIS
MESERVY, CLIFFORD JAMES, MEMPHIS
METZGER, WM EDGAR, MEMPHIS
MEYER, DAVID, MEMPHIS
* MILES, ROBT MILLARD, MEMPHIS
MILFORD JR, LEE WATSON, MEMPHIS
MILLER III, ROBERT HORACE, MEMPHIS
* MILLER JR, GEORGE L, MEMPHIS
* MILLER, FOUNTAIN FOX, MEMPHIS
MILLER, JOE HARDY, MEMPHIS
* MILLER, RICHARD ALVAH, MEMPHIS
MILLER, RICHARD B, MEMPHIS
* MILLER, RICHARD WALLACE, MEMPHIS
MILLER, THOMAS IVA, MEMPHIS
MILLS, GEO T, MEMPHIS
MILNOR III, JOHN PERVIS, MEMPHIS
MILNOR JR, J PERVIS, MEMPHIS
MINKIN, IRVING C, MEMPHIS
MIRVIS, DAVID MARC, MEMPHIS
- MITCHELL, BETH R, MEMPHIS
MITCHUM, WM ROBSON, MEMPHIS
* MOBLEY, EVERETT C, MEMPHIS
* MOELLER JR, BENJAMIN A, EADS
MOFFATT III, WILLIAM LEE, MEMPHIS
MOFFATT JR, WM LEE, MEMPHIS
* MOGAN, EDWARD NENON, GERMANTOWN
MOINUDDIN, MOHAMMED, MEMPHIS
MOINUDDIN, SHAMIM, MEMPHIS
MONAGHAN, THOMAS W, MEMPHIS
MUNGER JR, RALPH HORACE, MEMPHIS
MOORE JR, FONTAINE B, MEMPHIS
* MOORE JR, MOORE, MEMPHIS
* MOORE, DAVID F, MEMPHIS
MOORE, JAMES A, MEMPHIS
MOORE, MARION ROBERTSON, MEMPHIS
MOORE, MARSHA R, MEMPHIS
MORGAN, JACK COLBERT, MEMPHIS
MORISY, LEE RICHARD, MEMPHIS
MORRIS, JOHN THOS, MEMPHIS
MORRIS, WM RANDOLPH, GERMANTOWN
MORRISON, LARRY BURT, MEMPHIS
MORSE, JOHN CLAUDE, MEMPHIS
MORSE, WM HAL, MEMPHIS
- MORTIMER, JULIA A, MEMPHIS
MOSER, DAVIS D, MEMPHIS
MOSHIER, WM HILL, MEMPHIS
MOSKOVITZ, RONDALL J, MEMPHIS
* MOSS, JOHN PALMER, MEMPHIS
* MOSS, THOS CHESTER, MEMPHIS
MOSS, WM BENJ, MEMPHIS
MOTLEY, THOMAS EARL, MEMPHIS
MOUSTAFA, SALWA, MEMPHIS
MROZ, CHRISTINE T, MEMPHIS
MUHLBAUER, MICHAEL SCOTT, MEMPHIS
MUIRHEAD, ERNEST ERIC, MEMPHIS
MULLEN, JESSE G, MEMPHIS
MUNN, CHARLES W, MEMPHIS
MURDOCK, WADE THOS, MEMPHIS
* MURPHEY, FRANCIS, NAPLES, FL
MURPHY, CYNTHIA DABNEY, MEMPHIS
- MURPHY, GARNETT, MEMPHIS
MURPHY, JAMES GARNETT, MEMPHIS
MURPHY, PATRICK J, MEMPHIS
MURPHY, WALTER HENRY, MEMPHIS
MURPHY, WM MONT, MEMPHIS
MURRAH JR, WM FITZHUGH, MEMPHIS
MURRAY, IAN FARRELL, MEMPHIS
MYERS, WM STANLEY, MEMPHIS
- NABORS III, LOUIS B, MEMPHIS
NADEL, ALAN MARC, MEMPHIS
NASH, JOHN PAUL, MEMPHIS
NAWAF, KAYS, MEMPHIS
NEELY JR, CHAS LEA, MEMPHIS
- NELSON, MICHAEL W, MEMPHIS
NEWMAN, LARRY BERNARD, MEMPHIS
- NEWMAN, MARK S, MEMPHIS
NGUYEN, CHIEN, MEMPHIS
- NGUYEN, VINH DUC, MEMPHIS
NICHOLS, THOS WADDELL, MEMPHIS
NICHOPOULOS, GEORGE C, MEMPHIS
NIKOLOVSKI, OLIVER T, BIRMINGHAM, MI
NOBLES JR, EUGENE RODMAN, MEMPHIS
NOE, HORACE NORMAN, MEMPHIS
* NORMAN, ROBT SIDNEY, GERMANTOWN
- NORRIS, HUNTER WILLINGHAM, MEMPHIS
NORTH, WM C, MEMPHIS
NORTHERN JR, WM L, MEMPHIS
O'CONNELL, JOHN F, MEMPHIS
O'SULLIVAN, PATRICK JOS, MEMPHIS
OCHS, JEFFREY J, MEMPHIS
- OELLERICH, WILLIAM F, MEMPHIS
OGLE, EVELYN M BASSI, MEMPHIS
* OGLE, WM SANDERS, MEMPHIS
OGLESBY, CLAUDE DUNN, MEMPHIS
OKRAH, AMOS, MEMPHIS
* OLIM, CHAS BURTON, MEMPHIS
OLINGER, RODNEY GLENN, MEMPHIS
* ORMAN, JOS COOKE, MEMPHIS
ORPET JR, P E, MEMPHIS
OSBORN, FRANK JACKSON, MEMPHIS
OSBORNE, PAMELA THOMPSON, CORDOVA
- OSTEEN, ROBERT W, CORDOVA
OSWALD, WM J, MEMPHIS
OUTLAN, JOHN EDWARD, COLLIERVILLE
OUTLAN, WILLIAM F, COLLIERVILLE
OWENS, DONALD D, MEMPHIS
OWENS, JAMES HARVEY, MEMPHIS
* PACKER, HENRY, MEMPHIS
PAGE, GENE RUFFNER, MEMPHIS
PAGE, ROY C, MEMPHIS
PAIDIPALLI, BABU RAO, MEMPHIS
PAINTER, MAX WESLEY, MEMPHIS
- PAIS, WENDY J, MEMPHIS
- PALAZZO, ANTHONY J, MEMPHIS
PALMER IV, ROBERT E, MEMPHIS
PALMIERI, GENARO MIGUEL A, MEMPHIS
PARKER, JOS, MEMPHIS
PARKS, FRANK D, MEMPHIS
PARROTT JR, CHAS WM, SOUTHAVEN, MS
PARSONS III, WARD CHESTER, MEMPHIS
PARVEY, LOUIS S, MEMPHIS
PASLAWSKI, WALTER, MEMPHIS
* PASTER, SAML, SANTAMONICA, CA
* PASTERNACK, MORRIS, MEMPHIS
PATE, JAMES W, MEMPHIS
PATTERSON III, RUSSELL H, MEMPHIS
PATTERSON, CHARLES RICHARD, MEMPHIS
PATTERSON, KELLY, MEMPHIS
PATTERSON, SAM POLK, MEMPHIS
PATTERSON, STANLEY MARTIN, MEMPHIS
* PAUL, RAPHAEL NATHAN, MEMPHIS
PAYNE JR, EARNEST B, MEMPHIS
PAYNE, PAUL A, MEMPHIS
PEARSON, RICHARD MCQUISTON, MEMPHIS
PEDIGO, PHILLIP ADLER, MEMPHIS
PEELER, MOLLY M, MEMPHIS
PEEPLES JR, JOHN D, MEMPHIS
- PELLEGRIN JR, HENRI E, ARLINGTON
PENDER JR, JOHN VINCENT, MEMPHIS
PERRY, EDGAR EMRICH, MEMPHIS
PETERS, THOMAS GUY, MEMPHIS
PETTIT, PAUL N, MEMPHIS
PHELPS, WM CHAS, MEMPHIS
PHILLIPS, CAROL ANN, MEMPHIS
PHILLIPS, JERRY CLYDE, MEMPHIS
* PHILLIPS, WM EARL, MEMPHIS
PHOTOPULOS, GUY J, MEMPHIS
PIAN JR, MAURICE C, MEMPHIS
PIGOTT, JOHN D, MEMPHIS
PINSON, E LOUISE, MEMPHIS
PINSTEIN, MARTIN LEE, MEMPHIS
PITCOCK, JAMES ALLISON, MEMPHIS
- PITMAN, THOMAS C, MEMPHIS
PLATKIN, ALAN BAILEY, MEMPHIS
POLLNOW, ROBERT E, AMARILLO, TX
* POOL, ROBT MC CAUGHRIN, MEMPHIS
* PORTER, COLUMBUS HASSELL, MEMPHIS
PORTER, HUEY HENDERSON, MEMPHIS
PORTER, WM RICHARD, MEMPHIS
PORTERFIELD, JAMES G, MEMPHIS
POSEY, MICHAEL EVANS, MEMPHIS
POURCYROUS, MASSROO, MEMPHIS
POWELL, CARROLL E, MEMPHIS
PRASAD, SUDHA R, MEMPHIS
PRATT, THOMAS H, MEMPHIS
PRICE, CAROLYN CULPEPPER, MEMPHIS
PRICE, JAMES HOWARD, MEMPHIS
PRICE, ROBERT ALLEN, MEMPHIS
PRIDGEN, STEPHEN ALLEN, MEMPHIS
PRIDGEN, WM RUBY, MEMPHIS
PRIETO JR, LUIS CARLOS, MEMPHIS
PROCTOR, RUSSELL JAY, MEMPHIS
PRUITT, DAVID B, MEMPHIS
- PUGH, VIRGINIA ANN, MEMPHIS
- PURANIK, KANCHAN H, MEMPHIS
PUTMAN, BILLIE HAROLD, MEMPHIS
QUINN III, PETER JOS, MEMPHIS
RADA III, JOHN B, MEMPHIS
RAGHAVAIAH, N V, MEMPHIS
RAGSDALE, BLAKE, MEMPHIS
RAHMAN, MAHFUZUR, MEMPHIS
RAINES, EDWIN ALLEN, MEMPHIS
RAINES, RICHARD BROONAX, MEMPHIS
* RAINES, SAML LUCAS, MEMPHIS
* RAINEY, WM THOS, MEMPHIS
RAINS III, BOYCE MANKIN, MEMPHIS
RAMANATHAN, JAYA, MEMPHIS
RAMANATHAN, KUDANGUDI P, MEMPHIS
RAMEY III, DANL RANDOLPH, MEMPHIS
* RANDOLPH, JERRY F, MEMPHIS
RANDOLPH, PAUL DOUGLAS, MEMPHIS
RAO, BHASKAR NARAYAN, GERMANTOWN
RAWLINSON, WILLIAM T, MEMPHIS
RAWTANI, PALLAVI V, MEMPHIS
RAY, MORRIS WILLIAM, MEMPHIS
REAVES, EDWARD MC CORMICK, MEMPHIS
- REDDEN, R ALLEN, MEMPHIS
REED, CHESTON MURRAY, MEMPHIS
REED, EDWARD WILSON, MEMPHIS

REED, MARK LOYD, MEMPHIS
REEDER, ROBT CANADA, MEMPHIS
REESE JR, HARVEY C, MEMPHIS
REESE, HALDEN EUGENE, MEMPHIS
REISSER JR, JOHN MILTON, MEMPHIS
RENTROP, WALTER ANTON, MEMPHIS
RENTROP, WM EMIL, MEMPHIS
REYES, NORA V, MEMPHIS
REYNOLDS, GARY LYNN, MEMPHIS
RHEA JR, HAL S, MEMPHIS
RICHARDSON JR, ROBT LEE, MEMPHIS
- RICHARDSON, DAVID J, MEMPHIS
RICHARDSON, ELBERT GREER, MEMPHIS
RIGGS JR, WM WEBSTER, MEMPHIS
RIGGS, CHAS R, MEMPHIS
RILEY, FRANCES OSBORN, MEMPHIS
ROANE, JOURDAN ARCHIBALD, MEMPHIS
ROBBINS JR, SAMUEL GWIN, MEMPHIS
ROBBINS, EDWARD T, MEMPHIS
ROBERTS, LARRY K, MEMPHIS
ROBERTSON, JAMES THOS, MEMPHIS
ROBERTSON, JON HOBSON, MEMPHIS
- ROBERTSON, KENNETH R, MEMPHIS
* ROBINSON JR, CHAS G, MEMPHIS
ROBINSON JR, JOHN EDWARD, MEMPHIS
ROBINSON, JAMES A, MEMPHIS
ROBINSON, LLOYD EDWARD, MEMPHIS
ROBISON JR, LOWELL BENJ, MEMPHIS
ROCKETT, JOHN FREDERICK, MEMPHIS
- ROGERS, ALFRED H, MEMPHIS
* ROGERS, GORDON K, MEMPHIS
ROJAS, NORBERTO, MEMPHIS
ROLOSON, GARY JAMES, BARTLETT
RONEY, RONALD STEVEN, MEMPHIS
ROSE, DOUGLAS LYNN, MEMPHIS
ROSEN, GERALD MICHAEL, MEMPHIS
ROSENBERG, E WILLIAM, MEMPHIS
ROSENBERG, ZACHARY, MEMPHIS
ROSENSWEIG, JACOB, MEMPHIS
RODTT JR, WILLIAM EDWARD, MEMPHIS
RUCH JR, WALTER ALLWEIN, MEMPHIS
RUCH, ROBT MILTON, MEMPHIS
RUCKER, JAMES DANIEL, GERMANTOWN
* RUDNER JR, HENRY GORDON, MEMPHIS
RULEMAN, CHESTER ALLAN, MEMPHIS
RUNYAN JR, JOHN WM, MEMPHIS
RUSSELL JR, JOHN MURRAY, MEMPHIS
RUSSELL, THOMAS ANTHONY, MEMPHIS
RUSSO, WM LOUIS, MEMPHIS
RUTSCHMAN, JULIAN LEANDER, MEMPHIS
RYAN JR, GEO MARION, MEMPHIS
- RYU, JINA, MEMPHIS
SACHS, HANSON PHILIP, MEMPHIS
SACKS, HAROLD SAMUEL, MEMPHIS
SAELEY JR, CHAS FRANKLIN, MEMPHIS
SAGE, FRED P, MEMPHIS
SAINO, JAMES D, MEMPHIS
SALAZER, JORGE E, MEMPHIS
SALKY, NATHAN KALMON, MEMPHIS
SAMAHA, JOSEPH K, MEMPHIS
SAMMONS, LEHMAN CLARK, OSCEOLA, AR
SAMUELS, ALAN DANL, MEMPHIS
SANDER, CRAIG J, MEMPHIS
- SANDERS, B JEFF, MEMPHIS
SANDERS, FREDERICK D, MEMPHIS
- SANDERS, RODNEY C, MEMPHIS
* SANDERS, SAM HOUSTON, MEMPHIS
SANFORD JR, JACK CARTER, MEMPHIS
SANFORD, DAVID MARSHALL, MEMPHIS
SANFORD, ROBERT ALEXANDER, MEMPHIS
SATTERFIELD JR, WM T, MEMPHIS
SAUTER, ROBERT E, GERMANTOWN
SAXTON SR, GRADY L, GERMANTOWN
* SCHAFFER, DONALD EARL, MEMPHIS
SCHETTLER, BETTY J, MEMPHIS
SCHETTLER, WM HEYMOORE, MEMPHIS
SCHLESINGER, VICTOR ADLER, MEMPHIS
SCHMIDT, DONALD W, MEMPHIS
- SCHMIDT, WARREN N, MEMPHIS
SCHNAPP, MOACIR, MEMPHIS
SCHOETTLE JR, G PHILLIP, MEMPHIS
* SCHREIER, PHILLIP CHAS, MEMPHIS
SCHROEDER, HARRIETT L, LITTLE ROCK, AR
SCHWEITZER, JOHN B, MEMPHIS
SCHWERKOSKE, JOHN E, MEMPHIS
SCOTT III, BENJAMIN F, MEMPHIS
SCOTT JR, DANIEL J, MEMPHIS
SCOTT, EDWARD PATTON, MEMPHIS
SCOTT, EDWIN LEE, MEMPHIS
- SCOTT, HUGH BARRETT, MEMPHIS
SCOTT, JOS MANSON, MEMPHIS
- SCOTT, MARY LYNN, MEMPHIS
SCOTT, RANDALL LEE, MEMPHIS
SCRUGGS, JERRY L, MEMPHIS
SEALE, JAMES L, MEMPHIS
SEALS, JAMES L, MEMPHIS
SEBES, JENO IMRE, MEMPHIS
SEGAL, ANTHONY, MEMPHIS
SEGAL, JACK, MEMPHIS
* SEGAL, MAURICE P, MEMPHIS
* SEGERSON, EDWARD C, MEMPHIS
SEXTON, RAY OWEN, MEMPHIS
* SHAEFFER JR, S J, MEMPHIS
SHANKLIN, DOUGLAS R, MEMPHIS
- SHANKS JR, JAMES E, KNOXVILLE
SHAPIRO, MARVIN LOUIS, MEMPHIS
SHAPPLEY JR, WM VANCE, MEMPHIS
SHEA III, JOHN JOSEPH, MEMPHIS
SHEA JR, JOHN JOS, MEMPHIS
SHEA JR, MARTIN COYLE, MEMPHIS
SHEARIN, ROBT P N, MEMPHIS

* SHEFFIELD, WM E, MEMPHIS
SHELL III, DAN H, MEMPHIS
SHELTON, BRIXEY R, MEMPHIS
* SHELTON, JAMES R, HEBER SPRINGS, AR
SHELTON, THOMAS B, MEMPHIS
SHERROD II, ROME, MEMPHIS
SHIFFMAN, STEPHEN MURRAY, MEMPHIS
SHUMAKE, LESLIE BOWLIN, MEMPHIS
SHURLEY II, WILLIAM R, MEMPHIS
SIEGEL, BARRY ROSS, MEMPHIS
SIEGEL, JEROME SEYMOUR, MEMPHIS
* SIEGEL, SAUL, MEMPHIS
SIEVERS, RICHARD E, MEMPHIS
SIKES, JAMES C, MEMPHIS
SILVERMAN, MICHAEL N, MEMPHIS
- SIMCOX, MARK, BRISTOL
SIMMONS, JAMES C H, MEMPHIS
SIMS, CLIFFORD W, MEMPHIS
SINGLETON, MICHAEL ROBERT, MEMPHIS
SISK, THOS DAVID, MEMPHIS
* SISSMAN, PAUL R, MEMPHIS
SKAGGS, MARVIN RICHARD, MEMPHIS
SKAREDOFF, MICHAEL N, WILLOWBROOK, IL
* SKINNER, EDWARD HOLLAND, MEMPHIS
SLOAS, DAVID DALE, MEMPHIS
SLUTSKY, AVRON ABE, MEMPHIS
SMILEY, LINDA MARIE, MEMPHIS
* SMITH JR, HUGH MILBY A, MEMPHIS
SMITH JR, VERNON I, MEMPHIS
- SMITH, CAROL ANN, MEMPHIS
SMITH, CLYDE GAYLON, MEMPHIS
SMITH, KIRBY LEE, MEMPHIS
- SMITH, NELSON B, MEMPHIS
SMITH, STANLEY L, MEMPHIS
- SMITH, SULLIVAN R, COOKEVILLE
SMITH, VINCENT D, MEMPHIS
SMITH, W CHAPMAN, MEMPHIS
* SMYTHE JR, FRANK WARD, MEMPHIS
SNIDER, CHARLES VAN, MEMPHIS
SNYDER, DOWEN ERVIN, MEMPHIS
* SOHM, JOHN J, MEMPHIS
SOLOMITO, VINCENT LEE, MEMPHIS
SOLOWAY, MARK STEPHEN, MEMPHIS
SOPER, RICHARD GRAVES, MEMPHIS
- SPANN, MICHAEL G, MEMPHIS
SPENCER, JUDY, MEMPHIS
SPIOTTA JR, EUGENE J, MEMPHIS
SPIOTTA, EUGENE JOS, MEMPHIS
SPIOTTA, LARRY B, MEMPHIS
STANFORD, CARL COOPER, MEMPHIS
STANFORD, JAMES FRANKLIN, MEMPHIS
STANLEY JR, THOS V, MEMPHIS
* STARK, RAY GINGLES, MEMPHIS
STARR, JASON LEONARD, MEMPHIS
- STEIN, IRA E, MEMPHIS
STEIN, LEE S, MEMPHIS
STENTZ, DAVID L, MEMPHIS
STEPHENS, RAJ K, GERMANTOWN
* STEPP, WM PRICE, MEMPHIS
STERN, THOS NEWTON, MEMPHIS
STEVENSON, CLEO WILSON, MEMPHIS
* STEVENSON, EDWARD N, MEMPHIS
STEVENSON, ROBIN MALCOLM, MEMPHIS
- STEWART, GEORGE E, MEMPHIS
* STEWART, MARCUS JEFFERSON, MEMPHIS
STEWART, SHERRILL BRYCE, MEMPHIS
STOVALL, THOMAS GREGORY, MEMPHIS
STRASBERG, GARY DAVID, MEMPHIS
STRATTON, HENRY THOS, MEMPHIS
STROCK, SYLVIA S, MEMPHIS
STUBBLEFIELD, ROBT J, MEMPHIS
SULLIVAN, JAY MICHAEL, MEMPHIS
SULLIVAN, JOS ALBERT, MEMPHIS
- SUMMERS, D SCOTT, PARIS
SUMMITT, ROBERT LAYMAN, MEMPHIS
SUTHERLAND III, ARTHUR J, MEMPHIS
SUVILLAGA, VICTOR I, WILMINGTON, NC
SWEENEY, PATRICK JOHN, MEMPHIS
SYONOR, ELMER W, MEMPHIS
TABOR, OWEN BRITT, MEMPHIS
* TACKET, HALL SANFORD, MEMPHIS
TAG, ARNOLD K, MEMPHIS
* TALLEY, BYRON SHADRACH, MEMPHIS
TANENBAUM, MARK HARRIS, MEMPHIS
TAYLOR III, HERBERT A, MEMPHIS
TAYLOR JR, WM WOOD, MEMPHIS
TAYLOR, EDWIN OSCAR, MEMPHIS
TAYLOR, JOHN CHARLES, MEMPHIS
TAYLOR, MARTHA NEUMANN, MEMPHIS
* TAYLOR, ROBT CLARKE, MEMPHIS
- TAYLOR, SUSAN GRAY, MEMPHIS
TEAGUE, PAUL EDRO, MEMPHIS
TEJWANI, INDURANI A, MEMPHIS
TEMPLETON, TERRY P, MEMPHIS
TERHUNE, RONALD LYTLE, MEMPHIS
THOMAS JR, LLOYD R, GERMANTOWN
THOMAS, DIANNA J, MEMPHIS
- THOMAS, L BRANNON, EL JEBEL, CO
THOMPSON, BARRY E, MEMPHIS
THOMPSON, PAUL ANDREW, MEMPHIS
THOMPSON, TERRY L, MEMPHIS
- THOMPSON, TOM C, MEMPHIS
THOMPSON, TOMMY C, MEMPHIS
THOMSEN, WILLIAM B, MEMPHIS
THRELKELD, WM CLEAGE, MEMPHIS
THURMOND, S GAIL, MEMPHIS
TICKLE, SAML MILTON, MEMPHIS
TIELENS, DON RAYMON, MEMPHIS
TILLMAN, RONALD C, MOUNTAIN CITY
TODD, MARGARET RYAN, MEMPHIS
TODD, TANJA LU, MEMPHIS

TONKIN, ALLEN K, MEMPHIS
TONKIN, INA L D, MEMPHIS
TOOMS, ROBT EDWIN, MEMPHIS
TOSH, JOHN WILLIAMS, MEMPHIS
TOWNES, ALEXANDER S, MEMPHIS
- TRAN, PHONG HUNG, MEMPHIS
TRAPP, ERIKA F, MEMPHIS
TRAUTMAN, ROBERT J, MEMPHIS
TREADWELL III, GEORGE H, MEMPHIS
TREW, GARY F, MEMPHIS
* TRIPP, ALVIN BRUSH, NESBIT, MS
TROOP, BRYAN ROSS, MEMPHIS
TUBERVILLE, AUDREY WHALEY, MEMPHIS
* TULLIS JR, I FRANK, MEMPHIS
TULLIS, KENNETH FRANK, MEMPHIS
TURLEY III, JOHN C, MEMPHIS
* TURLEY JR, HUBERT KING, MEMPHIS
* TURLEY JR, JOHN C, MEMPHIS
TURMAN, PRENTISS A, MEMPHIS
TURNBULL JR, STEVE H, MEMPHIS
* TURNBULL, RANDOLPH R, MEMPHIS
TURNER, GEO RANDOLPH, MEMPHIS
TURNER, JAMES E, MEMPHIS
TURNER, JAN LEWIS, MEMPHIS
- TUTOR, DAVID K, MEMPHIS
* TYLER, LOUIS EDWARD, MEMPHIS
TYRER JR, AUSTIN ROY, MEMPHIS
* TYSON JR, WM T, MEMPHIS
UPSHAW, JAMES JERRY, MEMPHIS
UPSHAW, JEFFERSON DAVIS, MEMPHIS
USDAN, DAVID AARON, MEMPHIS
* VACCARO, EUGENE A, MEMPHIS
VARNER JR, C FERRELL, MEMPHIS
* VARNER, CLAUDE FERRELL, MEMPHIS
VARNER, JAMES CARROLL, MEMPHIS
VASU, RENGA I, MEMPHIS
VERA, SANTIAGO R, MEMPHIS
VERNER, WALTER EUGENE, MEMPHIS
VICK, EDWARD GRANT, MEMPHIS
VICK, SIDNEY D, MEMPHIS
VIERON, LEONIDAS NICHOLAS, MEMPHIS
VINCENT, JOHN ROBT, MEMPHIS
- VINCENT, KAREN ANN, MEMPHIS
VOOKLES, JOHN THORN, MEMPHIS
VU, TOLAN HOANG, GERMANTOWN
VU, TRONG VAN, MEMPHIS
WADE, W BURKE, MEMPHIS
- WAI, ROBERT B, MEMPHIS
- WAKEFIELD, PAUL, KNOXVILLE
* WARHAM, JAMES DALE, MADISON, MS
WALKER JR, PARKS W, MEMPHIS
WALKER, FRANCES CAROLYN, MEMPHIS
* WALKER, LILLIE C, LITTLE SWTZLAND, NC
WALKER, ROBERT A, MEMPHIS
WALKER, WILLIAM WHITE, MEMPHIS
* WALLACE, FRED C, MEMPHIS
* WALLACE, JAMES ASHFORD, MEMPHIS
* WALLACE, PETER B, MEMPHIS
WALZER, YAIR, MEMPHIS
WANDERMAN, RICHARD G, CORDOVA
WARDLAW, LEE LYLE, MEMPHIS
WARNER, RONNIE M, MEMPHIS
WARR III, OTIS SUMTER, MEMPHIS
* WARR, OTIS S, MEMPHIS
* WATKINS, WM W, MEMPHIS
WATRIDGE, CLARENCE B, MEMPHIS
WATSON, DONALD C, MEMPHIS
WATSON, SUSAN R, MEMPHIS
- WATTERS III, BRANDON, MEMPHIS
WEATHERLY, MARK WILLARD, MEMPHIS
WEBBER, BEN PORTER, MEMPHIS
WEBER III, ALVIN JULIAN, MEMPHIS
WEBER, BILL CARL, MEMPHIS
- WEDEGAERTNER, MARY ROSE, MEMPHIS
* WEEMS, JEROME J, MEMPHIS
WEEMS, JOS LELL, MEMPHIS
WEEMS, THOS DOYLE, MEMPHIS
- WEIL, MARTIN L, MEMPHIS
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- FAULKNER, LEE A, NASHVILLE
FAWCETT, KENNETH JAMES, NASHVILLE
FELCH, JAMES W, FRANKLIN
FELDMAN, RICHARD WARREN, NASHVILLE
FELTS, PHILIP W, NASHVILLE
FELTS, STEPHEN KAREY, HERMITAGE
FEMAN, STEPHEN S, NASHVILLE
FENICHEL, GERALD MERVIN, NASHVILLE
FERGUSON, HAROLD AUSTIN, NASHVILLE
* FESSEY, RAY D, NASHVILLE
FIELDS, JAMES P, NASHVILLE
FIELDS, JOHN PERSHING, NASHVILLE
FINCH, WILLIAM TYREE, NASHVILLE
FINKE, FREDERICK LEROY, NASHVILLE
FISHBEIN, JOS H, NASHVILLE
FISHER, BENJ, NASHVILLE
FISHER, JACK, NASHVILLE
* FISHER, LAURA M, NASHVILLE
FLEET JR, WILLIAM F, GOODLETTSVILLE
FLEISCHER, ARTHUR C, NASHVILLE
FLEMING JR, JAMES H, NASHVILLE
FLEMING JR, ROSS, NASHVILLE
FLEMING, PHILIP EDWARD, NASHVILLE
FLETCHER, JOHN RAYMOND, NASHVILLE
FLETCHER, SUZANNE M, MADISON
FLEXNER, JOHN MORRIS, NASHVILLE
FOLEY, GERALD J, WHITE BLUFF
FOREMAN, HOWARD R, NASHVILLE
FORSYTH, G PAUL, NASHVILLE
FOSTER, HENRY WENDELL, NASHVILLE
* FOSTER, JOHN H, NASHVILLE
FOSTER, NELSON RAY, FRANKLIN
FOWINKLE, EUGENE WESLEY, NASHVILLE
* FOWLER, SAML B, NASHVILLE
FRANCIS, ROBT STANLEY, NASHVILLE
FRANKLIN, JERRY M, NASHVILLE
FREDERIKSEN, RAND TERRELL, NASHVILLE
FREEMAN, RUFUS JACK, NASHVILLE
FRENCHMAN, KHUSHRU H, HENDERSONVILLE
FREY, WALTER WILLIS, NASHVILLE
FRIDDELL, THOS JAMES, NASHVILLE
FRIESINGER, GOTTLIEB C, NASHVILLE
FRIST JR, JOHN C, NASHVILLE
FRIST, ROBT ARMISTEAD, NASHVILLE
* FRIST, THOS F, NASHVILLE
FRIST, WILLIAM HARRISON, NASHVILLE
FURMAN, JOHN ROBERT, MADISON
GAINES, DONALD LEE, NASHVILLE
* GANT, JULIAN C, LOMA LINDA, CA
* GARDNER, CHAS KURTIN, NASHVILLE
GARMAN, RICHARD W, BRENTWOOD
GAROVICH, MICHAEL C, NASHVILLE
* GARRETT, SAM YOUNG, NASHVILLE
GASKINS, FAY M, NASHVILLE
GASTON JR, ROBERT B, NASHVILLE
GASTON, ROBT B, DONELSON
GAVIGAN, WM MITCHEL, NASHVILLE
GAW, DAVID WISDOM, NASHVILLE
GEDDIE, DANL CLARK, NASHVILLE
GENCA, EROL, NASHVILLE
GENTILE, DOUGLAS A, NASHVILLE
GENTRY, HAROLD LEFFEL, MADISON
* GESSLER, CARL NEWTON, NASHVILLE
GHOSH, SUDHIR C, BRENTWOOD
GIBSON, JOHN RAGAN, NASHVILLE
GIBSON, RICHARD L, NASHVILLE
GILL, CHAS MC CLELLAND, NASHVILLE
- GILLIAM, DAVID MARK, NASHVILLE
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- GIVENS, TIMOTHY G, CINCINNATI, OH
GLASCOCK, FRANK B, NASHVILLE
GLASSCOCK, MICHAEL F, NASHVILLE
GLASSFORD JR, DAVID M, NASHVILLE
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GLICK, ALAN DOUGLAS, NASHVILLE
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GLUCK JR, FRANCIS W, NASHVILLE
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GOLDNER JR, FRED, NASHVILLE
* GOMEZ, PAUL CHAS, NASHVILLE
GOODMAN, BRUCE RANDOLPH, NASHVILLE
GOODMAN, WILLIAM M, MADISON
GORE, JOHNNY ELMO, ANTIOCH
GORSTEIN, FRED, NASHVILLE
GOWDA, HIRANYA C K, NASHVILLE
GRABER, ALAN LEE, NASHVILLE
GRAHAM JR, LOUIS S, NASHVILLE
GRAHAM JR, ROBERT P, NASHVILLE
GRAHAM, THOMAS P, NASHVILLE
GRANDA, ANTONIO MEDARDO, NASHVILLE
GRANT, BURTON PAINE, NASHVILLE
GRAVES JR, HERSCHEL A, NASHVILLE
GRAY, GEORGE F, NASHVILLE
GRAY, ROLAND WILLIAM, NASHVILLE
GRECO, FRANK ANTHONY, NASHVILLE
GREEN JR, PAUL A, NASHVILLE
GREEN, EDMON LEE, NASHVILLE
GREEN, JAMES DONALD, NASHVILLE
- GREEN, KELLI C, NASHVILLE
* GREEN, LOUIS D, BRENTWOOD
GREEN, NEIL EDWARD, NASHVILLE
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GREENE, HARRY LEE, NASHVILLE
GREENE, JOHN W, NASHVILLE
GREER JR, CLIFTON E, NASHVILLE
GREER, JOHN PETTRY, NASHVILLE
GREGG, CLARK R, NASHVILLE
GREGG, SUSAN BRITTINGHAM, NASHVILLE
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GREGORY, DAVID WILSON, NASHVILLE
GREMILLION JR, DANIEL E, NASHVILLE
GRIFFIN, JOHN JOS, NASHVILLE
GRINDE, STEPHEN E, NASHVILLE
GRINER, WINSTON H, NASHVILLE
GRISCOM, JOHN HOOPER, NASHVILLE
GROOS, ERICH BRYAN, NASHVILLE
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* GROSSMAN, MILTON, NASHVILLE
GROVE, R BARRY, BRENTWOOD
GROWDON JR, JAMES HAROLD, NASHVILLE
GUILLERMIN, JOHN PHILIP, HERMITAGE
GUNN, MICHAEL G, NASHVILLE
GURLEY, LARRY D, NASHVILLE
GUTOW, GARY SAML, NASHVILLE
GUTOW, RICHARD FINEMAN, NASHVILLE
HAGAN, GEO BRYANT, MADISON
HAGAN, KEITH W, NASHVILLE
HAGAN, KEVIN F, NASHVILLE
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HAINSWORTH, JOHN D, NASHVILLE
HALEY JR, ROBT LEO, MADISON
HALL JR, WALLACE HOWARD, NASHVILLE
HALL, HUGH DAVID, NASHVILLE
HALTOM, THOS BRANSON, NASHVILLE
HAMBERG, MARCELLE ROBT, NASHVILLE
HAMBURGER, NORMAN J, BRENTWOOD
HAMILTON, CHAS M, NASHVILLE
HAMILTON, JAMES RICHARD, NASHVILLE
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HAMMONDS, ROY GLENN, NASHVILLE
HANOTE, ROBERT E, BRENTWOOD
HANES, THOMAS EUGENE, MADISON
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HARDIN, ROBT ALLEN, NASHVILLE
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HARRIS, JEFFREY S, NASHVILLE
HARRIS, PERRY FELTON, NASHVILLE
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HAWKINS, ROWLAND SPECK, NASHVILLE
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HAYNES JR, J BREVARD, NASHVILLE
HAYNES, JAMES HUGH, NASHVILLE
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HAYS, JAMES WM, NASHVILLE
HEFLIN, A CLYDE, NASHVILLE
HEIM, CRAIG REED, NASHVILLE

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HELME, JAMES B, NASHVILLE
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HESTER, RAY WILLIS, NASHVILLE
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HILL, DAVID EDWIN, NASHVILLE
HILL, WARREN THOS, MADISON
HILL, WM HAROLD, MADISON
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HILLS, EDWARD RUDOLPH, NASHVILLE
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HIRSHBERG, CHAS SNYDER, NASHVILLE
HITCHMAN, JAMES KENNETH, NASHVILLE
HOBDY, CHARLIE JOE, NASHVILLE
HOLCOMB JR, GEO W, NASHVILLE
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HOLLIFIELD, JOHN WARD, NASHVILLE
HOLMES III, GEO LANDIS, NASHVILLE
HOLZEN, THOMAS W, NASHVILLE
HOOD, ROB REID, NASHVILLE
HOOS, RICHARD T, NASHVILLE
HORN, ROBT GORDON, NASHVILLE
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HSUEH, YERNG TERNG, NASHVILLE
HUBER, THOS J, MADISON
HUDGINS, JAMES M, HENDERSONVILLE
HUFFMAN, WILLIAM R, BRENTWOOD
HUGGINS, TERESA G, MURFREESBORO
HUMPHREY, STEPHEN P, MADISON
HUMPHREYS, JERRY KAY, MT JULIET
HUNT, JERRY CHEEK, MADISON
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HUSTON, JOSEPH W, NASHVILLE
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IKARD, ROBT WINSTON, NASHVILLE
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* ISMAIL, MOHAMMED, NASHVILLE
* IVIE, JOS MC KINNEY, NASHVILLE
JACKSON, C GARY, NASHVILLE
JACKSON, ROGER THEODORE, NASHVILLE
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JAMES, A EVERETTE, NASHVILLE
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JENNINGS, HENRY S, NASHVILLE
JERKINS, GARY W, NASHVILLE
JERKINS, TERRI WOOD, NASHVILLE
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JOHNSON JR, IRA T, NASHVILLE
JOHNSON, DAVID HORTON, NASHVILLE
JOHNSON, HARRY KEITH, NASHVILLE
* JOHNSON, HOLLIS EUGENE, NASHVILLE
JOHNSON, JAMES WM, NASHVILLE
JOHNSON, JOHN SETTLE, NASHVILLE
JOHNSON, PAUL ALFRED, NASHVILLE
JOHNSON, ROBT MARSHALL, NASHVILLE
JOHNSTON, CHAMBLESS RAND, NASHVILLE
JOHNSTON, ROBT K, NASHVILLE
JOHNSTON, WILLIAM D, NASHVILLE
JONES III, HOWARD W, NASHVILLE
JONES JR, ORRIN LESTER, NASHVILLE
JONES, BRUCE E, NASHVILLE
JONES, DAVID NANDO, NASHVILLE
JONES, DAVID SCOTT, NASHVILLE
JONES, EDMUND PALMER, NASHVILLE
JONES, FRANK EMERSON, NASHVILLE
JONES, JOHN DONALD, NASHVILLE
JONES, PHILLIP R, NASHVILLE
* JORDAN, THOS MALONE, NASHVILLE
JULIAO, SAUL A, NASHVILLE
JUSTICE JR, PAUL ANDREW, NASHVILLE
KAISER, ALLEN B, NASHVILLE
KAMBAM, JAYAKUMAR REDDY, NASHVILLE
KAMINSKI, MICHAEL JAMES, NASHVILLE
* KAMPMEIER, RUDOLPH H, NASHVILLE
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KAPLAN, PETER ROBT, NASHVILLE
KARZON, DAVID THEODORE, NASHVILLE
KASSELBERG, ALFRED GUY, NASHVILLE
KAY, JENNIFER L, NASHVILLE
KAYE, JEREMY JON, NASHVILLE
KEANE, WM SHERMAN, NASHVILLE
KEMMERLY, PAUL COURTLAND, NASHVILLE
* KENDALL, CYRUS ERVE, HENDERSONVILLE
* KENNEDY, J ALLEN, NASHVILLE
KENNER III, WM DAVIS, NASHVILLE
* KENNON JR, WM GILLIAM, NASHVILLE
KEOWN, MARY ELIZABETH, NASHVILLE
KEYSER III, JOHN EDWARD, NASHVILLE
KHAN, QAMAR ALI, NASHVILLE
KILLMAN, KATHRYN R, NASHVILLE
KILROY, ANTHONY WALDO, NASHVILLE
- KIM, ALEX H, NASHVILLE
KIMBRELL JR, FRED TAYLOR, DONELSON
KINCAID, ROBERT SAMUEL, FRANKLIN
KING JR, LLOYD E, NASHVILLE
KINNARD, JOHN PARKES, NASHVILLE
KINNEY, STEVEN R, NASHVILLE
KIRBY, LOWRY DALE, NASHVILLE
KIRCHNER JR, FREDERICK K, NASHVILLE
KIRCHNER, SANDRA LYNNE G, NASHVILLE
KIRSHNER, HOWARD S, NASHVILLE
KLING JR, RALPH R, NASHVILLE
KOCH, MICHAEL D, NASHVILLE
KOCHTITZKY, OTTO M, NASHVILLE
KOENIG, LEONARD J, NASHVILLE
KOOMEN, JOHN C, NASHVILLE
KOURANY, RONALD FREDERIC, NASHVILLE
* KRAMER, LEE F, GOODLETTSVILLE
KRONENBERG, MARVIN W, NASHVILLE
KUZUR, MICHEL ELIAS, NASHVILLE
KYGER, KENT, NASHVILLE
* LA VOI, SAML JOS, NASHVILLE
LADD, JAMES DUNCAN, NASHVILLE
- LAGRONE, ROBERT P, NASHVILLE
LAMB, JOHN WM, NASHVILLE
* LAMB, ROLAND D, NASHVILLE
LAMBALLE, ADRIAN K, NASHVILLE
LAMBERT, FRANK HAYDEN, NASHVILLE
LANDMAN, JEFFREY A, MADISON
- LAROCCA, CHRISTINE D, NASHVILLE
LATOUR, DANA L, FRANKLIN
LATOUR, PAUL A, NASHVILLE
LAUGHLIN, LAWRENCE PAUL, NASHVILLE
LAVELY JR, HORACE T, NASHVILLE
* LAWRENCE JR, GRANVILLE A, NASHVILLE
LAWS, KENNETH HOWARD, NASHVILLE
LAWSON, ALBERT ROBT, MURFREESBORO
LE QUIRE, VIRGIL S, NASHVILLE
LEA IV, JOHN W, NASHVILLE
LEA, CLARK D, NASHVILLE
LECORPS, PATRICK J, FRANKLIN
LEDBETTER, WILLIAM HENRY, NASHVILLE
LEE, DAVID GRANVILLE, NASHVILLE
LEEPER, HOWARD B, BRENTWOOD
LEFKOWITZ, LEWIS B, NASHVILLE
LEFTWICH, RUSSELL B, NASHVILLE
LENTZ, JOS FRANCIS, NASHVILLE
LEONARD, JOHN MARTIN, NASHVILLE
LESTER, JAMES PEYTON, NASHVILLE
LEVITT, MICHAEL J, NASHVILLE
LEWIS, MALCOLM R, NASHVILLE
* LIDDLE, GRANT WINDER, NASHVILLE
LIGHT, RICHARD T, NASHVILLE
LILLY, EDWIN JACOB, NASHVILLE
LIMBIRD, THOMAS J, NASHVILLE
LINK, JOHN LOUIS, NASHVILLE
LINN, JOANNE LOVELL, NASHVILLE
LINN, ROBT J, NASHVILLE
LIPSCOMB JR, ALBERT BRANT, NASHVILLE
LIPSCOMB, ALBERT BRANT, NASHVILLE
LISELLA, RICHARD SCOTT, NASHVILLE
LITTLE, JAMES P, CEDAR HILL
LLOYD, KENNETH MICHAEL, NASHVILLE
LODEN, JAMES POPE, NASHVILLE
LONG, RUTH BARRON, NASHVILLE
LONG, WM ROYSTON, NASHVILLE
- LOPATINE, STEVEN M, NASHVILLE
LOVELACE, DONALD RAY, NASHVILLE
LOVEN, KEITH H, NASHVILLE
LOVVORN JR, HAROLD N, NASHVILLE
- LOWERY III, DOUGLAS W, ST PETERSBURG, FL
LUBOW, LAWRENCE D, NASHVILLE
LUNDIN, LINDA S, NASHVILLE
* LYLE, PHILIP LEWIS, NASHVILLE
LYNCH, JOHN BROWN, NASHVILLE
MACIUNAS, ROBERT J, NASHVILLE
MACMILLAN JR, CHAS W, NASHVILLE
MACMILLAN, ROBT DUNCAN, NASHVILLE
MADDEN JR, JAMES JOS, BRENTWOOD
MAGEE, MICHAEL J, NASHVILLE
MAGPANTAY, EDMUNDO D, MT JULIET
* MAGRUDER, ROBT HERMAN, OLD HICKORY
MALLARD, ROBT ELWOOD, NASHVILLE
MANALAC SR, ABELARDO Z, MT JULIET
MANNING, DEBORAH A, NASHVILLE
MARNEY, SAMUEL ROWE, NASHVILLE
MARTIN III, RAYMOND S, NASHVILLE
MARTINEZ, ERLINDA A, NASHVILLE
MARTINEZ, ROGELIO R, NASHVILLE
MASON, THOS EMMETT, NASHVILLE
MASSIE, RALPH W, NASHVILLE
MAXWELL, G PATRICK, NASHVILLE
* MAYER, JAMES A, NASHVILLE
* MAYES, BEN RICHARDSON, NASHVILLE
MAYES, CHAS EUGENE, NASHVILLE
MCALISTER, AILEEN HOOD, NASHVILLE
MCCALL, HERBERT TRAVIS, MADISON
MCCLELLAN, ROBERT E, NASHVILLE
MCCLELLAN, ROBERT TRIGG, NASHVILLE
MCCOMBS, PAUL RAYMOND, NASHVILLE
MCCONNELL, CONN M, MADISON
* MCCRACKEN, ROBERT LAZEAR, NASHVILLE
MCDONALD, EDWARD C, NASHVILLE
MCDOUGAL, WILLIAM S, NASHVILLE
MCFERRIN, JAMES R, NASHVILLE
MCGEHEE, JAMES BARTLEY, NASHVILLE
MCGINLEY, JAMES HENRY, NASHVILLE
MCGINNIS, CHARLES W, NASHVILLE
MCGREW, WALLACE, NASHVILLE
MCINNIS, JOHN CAMERON, NASHVILLE
MCKAY, CHARLES E, NASHVILLE
MCKEE, DAVID EARL, MADISON
MCKENNA, SAMUEL JAY, NASHVILLE
MCLEOD, ALEXANDER C, NASHVILLE
MCMAHAN, JOHN WELLINGTON, NASHVILLE
MCMURRAY, M CHARLES, NASHVILLE
MCMURTRY, CECIL E, MADISON
MCNABB, PAUL CARTER, NASHVILLE
MCPHERSON, EWING WILLIAM, NASHVILLE
MCPHERSON, WARREN E, NASHVILLE
MCQUEEN, SAMUEL, HERMITAGE
MCRAE, JOHN RADFORD, NASHVILLE
MEACHAM, PATRICK W, NASHVILLE
* MEACHAM, WM FELAND, NASHVILLE
MEADOR, CLIFTON K, NASHVILLE
MEADORS, MICHAEL H, NASHVILLE
MELKIN, STEPHEN PELLAR, NASHVILLE
MENDOZA, DANIEL, HENDERSONVILLE
MENZIE, JAMES W, NASHVILLE
MERRILL, WALTER WILSON, NASHVILLE
MERRITT II, CULLEN R, NASHVILLE
METTS III, VERGIL L, MADISON
MEYER JR, ALVIN HENRY, DONELSON
MICHAEL, PAUL R, NASHVILLE
MILEK, MICHAEL A, NASHVILLE
MILLER JR, JAMES OLNEY, MADISON
MILLER, ANDREW HERRON, NASHVILLE
MILLER, BONNIE MERSKY, NASHVILLE
MILLER, DAVID STUART, NASHVILLE
MILLER, JOE M, NASHVILLE
MILLER, JOHN M, NASHVILLE
* MILLER, LLOYD C, NASHVILLE
MILLER, MICHAEL E, NASHVILLE
MILLER, MICHAEL PETER, NASHVILLE
- MILLER, THEODORE T, NASHVILLE
MILLER, THOMAS B, MADISON
MILLIS, JAMES BROWN, NASHVILLE
MINCH, F MICHAEL, NASHVILLE
* MINTON, LEE ROY, NASHVILLE
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MITCHELL, CARL EDWARD, NASHVILLE
MITCHELL, CHARLES AUSTIN, NASHVILLE
MITCHELL, DOUGLAS PARK, NASHVILLE
MITCHELL, EDWIN HARRIS, NASHVILLE
MITCHELL, LARRY M, NASHVILLE
MITCHUM, ALBERT JACKSON, CLARKSVILLE
MODJESKA, GERALD S, NASHVILLE
MOGAN, THOS FRANCIS, HENDERSONVILLE
MOLIN, JOHN A, NASHVILLE
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- MONROE, JAMIE MARIA, NASHVILLE
MONTGOMERY, DEBORAH G, NASHVILLE
MONTGOMERY, MARCIA A, NASHVILLE
MONTOURIS, GEORGIA D, NASHVILLE
MOORE, JAMES N, NASHVILLE
MORAN, HOUSTON, NASHVILLE
MOREHEAD, V TUPPER, NASHVILLE
MORGAN, DAVID H, NASHVILLE
MORONEY, DAVID M, NASHVILLE
MORRIS JR, JOHN A, NASHVILLE
MORTON III, CHARLES F, NASHVILLE
MOULTON, PATRICK HOWARD, NASHVILLE
MOYERS, JAMES RICHARD, NASHVILLE
MULHERIN JR, JOS LOUIS, NASHVILLE
MURRAY JR, ROBERT C, NASHVILLE
- MURRAY, ELIZABETH ANN, NASHVILLE
* MURRAY, HENRY DARWIN, OLD HICKORY
NACE, GARY STEPHEN, NASHVILLE
NADEAU, JOHN HUGH, NASHVILLE
NAJJAR, JENNIFER LEE, NASHVILLE
NASH, JAMES L, NASHVILLE
NEADERTHAL, ROBERT LEE, NASHVILLE
NEBLETT III, WALLACE W, NASHVILLE
NEFF, BETTY K, NASHVILLE
NELSON, DUNKIN AFTON, NASHVILLE
NELSON, I ARMISTEAD, NASHVILLE
NELSON, LOREN DOUGLAS, NASHVILLE
NEMEC, DEWEY G, NASHVILLE
NESBITT JR, THOMAS E, NASHVILLE
NESBITT, TOM EDWARD, NASHVILLE
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NETTERVILLE JR, JOHN T, BRENTWOOD
NEUBAUER, STEVEN W, NASHVILLE
NEWSOME III, H CLAY, NASHVILLE
NICHOLS, DARYL L, NASHVILLE
NIEDERMEYER, MICHAEL E, NASHVILLE
NOEL JR, PHILIP JORDAN, NASHVILLE
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NORRIS, MARGARET SWANN, NASHVILLE
NORTH, K TIMOTHY, MADISON
NORTON, CHAS GLENN, NASHVILLE
NORTON, DOUGLAS EDWARD, NASHVILLE
NUNN, PAULA S, NASHVILLE
NYLANDER, BARBARA HARTKOP, NASHVILLE
NYLANDER, WILLIAM ARTHUR, NASHVILLE
O'BRIEN, KEVIN MICHAEL, MADISON
O'DAY, DENIS MICHAEL, NASHVILLE
OATES, JOHN ALEXANDER, NASHVILLE
OGLESBY, JOHN WILLS, NASHVILLE
OLDHAM, RICHARD RANDOLPH, NASHVILLE
OLLAPALLY, ELSIE P, MADISON
OLSEN, DOUGLAS OLE, BRENTWOOD
OLSON, BARBARA JEAN, NASHVILLE
ONI, ADEDAMOLA, NASHVILLE
ORCUTT, THOS WM, NASHVILLE
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ORTH, DAVID N, NASHVILLE
OSSOFF, ROBERT H, NASHVILLE
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OVERFIELD, RONALD EDWIN, NASHVILLE
OWEN, ROBT CARROLL, NASHVILLE
OWNBEY, RICHARD PHILLIP, NASHVILLE

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PAGE JR, HARRY L, NASHVILLE
PAGE, DAVID LEE, NASHVILLE
PALACIO DEL VALLE, GUSTAVO, NASHVILLE
PANLILIO, ADELISA, NASHVILLE
PARANJAPE, YESHAWANT B, HERMITAGE
PARDUE, CHRIS C, NASHVILLE
PARKER, ROY W, NASHVILLE
PARRIS, WINSTON CLIVE VIC, NASHVILLE
PARRISH, CAROLYN M, NASHVILLE
PARRISH, THOS FRANKLIN, NASHVILLE
PARTAIN, C LEON, NASHVILLE
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• PASS, BERNARD J, NASHVILLE
PATE, JOHN KIRBY, NASHVILLE
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PATTEN, W THOMAS, MADISON
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